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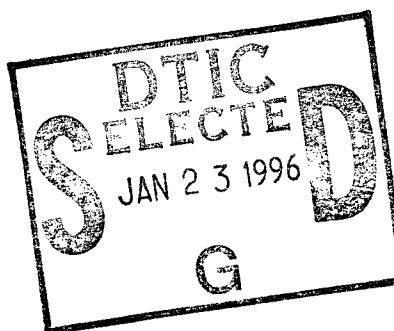
RAISING THE OPERATIONAL CEILING: A WORKSHOP ON THE LIFE SUPPORT AND PHYSIOLOGICAL ISSUES OF FLIGHT AT 60,000 FEET AND ABOVE

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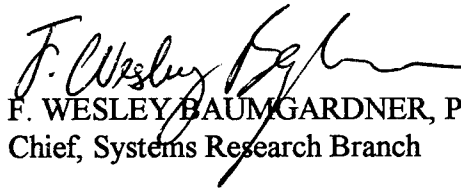**BROOKS AIR FORCE BASE, TEXAS**

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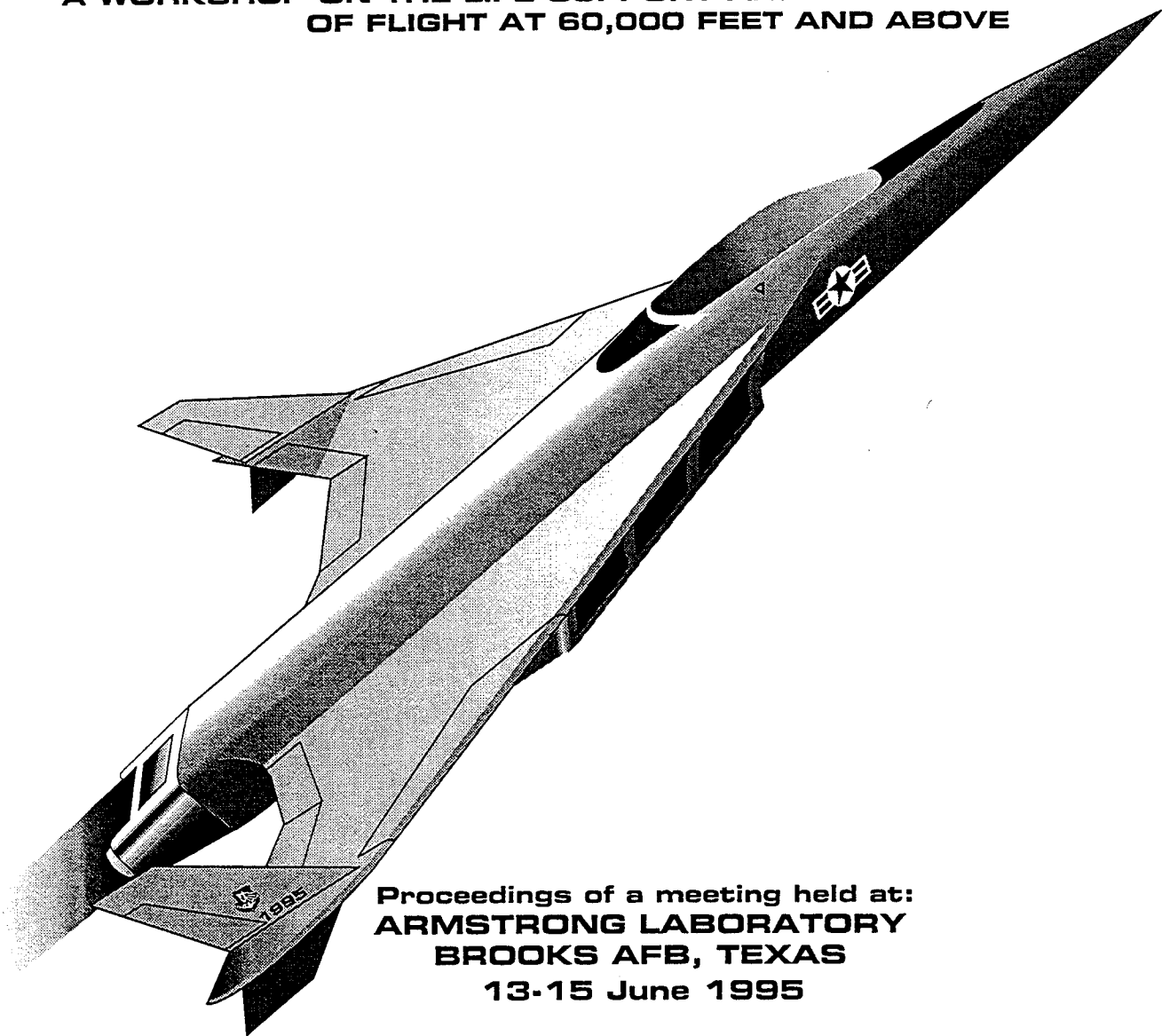
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RAISING THE OPERATIONAL CEILING

A WORKSHOP ON THE LIFE SUPPORT AND PHYSIOLOGICAL ISSUES OF FLIGHT AT 60,000 FEET AND ABOVE

Proceedings of a meeting held at:
ARMSTRONG LABORATORY
BROOKS AFB, TEXAS
13-15 June 1995

Co-Chaired and Edited by
Andrew A. Pilmanis Ph.D.
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The altitude ceiling for selected high performance aircraft, e.g., the F-22 and Eurofighter 2000 has been raised from 50,000 to 60,000 feet. The impact of this change is complex and could impose decisive limitations with regard to crew safety issues. Detailed exposure limits, specifications and standards are required for the development of life support equipment, operational procedures, plans and training programs for exposure to these high altitude conditions. Developing these limits, specifications and standards requires a comprehensive research database for effective solutions.

Over forty key individuals were brought together for a three day workshop at the Armstrong Laboratory, Brooks AFB, Texas on 13 to 15 June 1995, to discuss, coordinate and provide initial input for tracking life support interrelationships which might have the potential to evolve into an operational limits database. The objectives of the workshop were to encourage direct discussion across disciplines by: 1) outlining the rationale for operating at higher altitudes as well as current life support equipment capabilities under development; 2) reviewing and integrating the physiological and life support requirements for increasing the operational altitude; 3) establishing limitations, additional research and equipment requirements and trade studies and; 4) providing a timely publication covering relevant issues to prevent crew safety from becoming a limiting factor in future high altitude operations.

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Abbreviations and Acronyms

ABS	Aviators Breathing System
ACC	Air Combat Command
AFB	Air Force Base
AFI	Air Force Instruction
AGARD	Advisory Group for Aerospace Research and Development
AL	Armstrong Laboratory
ASCC	Air Standardization Coordinating Committee
ATAGS	Advanced Technology Anti-G Suit
AVO2	Arterial Venous Oxygen
AWACS	Airborne Warning and Control System
BOS	Backup Oxygen System
CNS	Central Nervous System
CO2	Carbon Dioxide
COMBAT ACE	Combined Advanced Technology Acceleration/Chemical Ensemble
COMBAT EDGE	Combined Advanced Technology Enhanced Design G Ensemble
DACT	Disposable Absorption Containment Trunk
DCIEM	Defense and Civil Institute of Environmental Medicine
DCS	Decompression Sickness
ECS	Environmental Control System
FEBA	Forward Edge of the Battle Area
FIO2	Fraction of Inspired Oxygen
FRC	Functional Residual Capacity
G-LOC	G-induced Loss of Consciousness
+Gz	Head-to-foot inertial force
HSC	Human Systems Center
IOP	Initial Operating Procedure
IOT&E	Inflight Operational Test and Evaluation
LEP	Laser Eye Protection
MILSPEC	Military Specification
MSOC	Molecular Sieve Oxygen Concentrator
MSOGS	Molecular Sieve Oxygen Generating System
OBOGS	On Board Oxygen Generating System
PAO2	Alveolar Partial Pressure of Oxygen
PBA	Positive Pressure Breathing for Altitude
PBG	Positive Pressure Breathing for G
PCO2	Partial Pressure of Carbon Dioxide
PO2	Partial Pressure of Oxygen
PPB	Positive Pressure Breathing
PSI	Pounds per Square Inch
PSID	Pounds per Square Inch Differential
RAF	Royal Air Force
SAM	Surface to Air Missile
SPO	Systems Program Office
TLSS	Tactical Life Support System
UCD	Urine Collection Device
UK	United Kingdom
US	United States
USAF	United States Air Force
USC	University of Southern California
USN	United States Navy
UWARS	Under Water Active Release System
VGE	Venous Gas Emboli

Preface

The altitude ceiling for selected high performance aircraft, e.g., the F-22 and Eurofighter 2000, has been raised from 50,000 to 60,000 feet. The impact of this change is complex and could impose decisive limitations with regard to crew safety issues. Detailed exposure limits, specifications, and standards are required for the development of life support equipment, operational procedures, plans, and training programs for exposure to these high altitude conditions. Developing these limits, specifications, and standards will require a comprehensive research database for effective solutions.

A multitude of physiological and life support aspects of high-altitude flight have been examined by experts in various specialty areas, such as aircraft pressurization, oxygen and pressure breathing systems, cardiopulmonary effects and decompression sickness. However, much of the current database involves extrapolation from earlier studies at lower altitudes or low pressure chamber studies that have not fully taken into account the operational flight exposure conditions. One such example is a loss of cabin pressure after exposure to cabin altitudes of 22,500 feet for a period of time. With a rapid decompression to 60-65,000 feet, little solid data exists to decide the lower altitude to which the aircrew member should descend for continuing the flight. A comprehensive and integrated approach is necessary to ensure that crew safety is not compromised by these operational changes.

To maintain the operational safety of aircrew exposed to high altitude conditions, there must be an understanding of both the subtle and profound, as well as the short- and long-term effects resulting from the exposure. Much of the rich scientific database on exposure to high altitude has been reported at annual meetings and in journals. Because of publication delays and limited attendance at the meetings, these reporting methods are often inadequate for disseminating information.

The most effective way to bring the life support community to a comprehensive understanding of current research, as well as gaps in the knowledge base, was to bring together an expert panel and conduct a workshop on the life support and physiological issues of flight at 60,000 feet. The objectives of the workshop, therefore, were to encourage direct discussion across disciplines by:

1. Outlining the rationale for operating at higher altitudes, as well as current life support equipment capabilities under development.

2. Reviewing and integrating the physiological and life support requirements for increasing the operational altitude.

3. Establishing limitations, additional research and equipment requirements and trade studies.

4. Providing a timely publication covering relevant issues to prevent crew safety from becoming a limiting factor in future high altitude operations.

Over forty key individuals were brought together for a three-day workshop at the Armstrong Laboratory, Brooks AFB, Texas, on 13 to 15 June 1995, to discuss, coordinate, and provide initial input for tracking life support interrelationships that might potentially evolve into an operational limits database. The meeting was conceived as a workshop, not a scientific symposium. The emphasis was placed on open discussion and the exchange of information. The papers were intended to be topic reviews rather than original scientific presentations. Their purpose was to establish the basis for further discussion and to update the participants on current programs involving high altitude and life support equipment. The design of the workshop included not only scientific fact, but opinion and conjecture as well.

The products from the workshop include a special report of the proceedings, a compendium of research and development data, variables relating to the various aspects of raising the ceiling, and a summary of recommendations.

EXECUTIVE SUMMARY

Dramatically improved airframe technology and the extreme mission environments of modern air warfare threaten to overwhelm the physiological and cognitive capacities of aircrew. The problem is magnified when increased systems capabilities are considered in the context of the potency and density of weapons possessed by potential adversaries. It is clear that today's mission requirements and sophisticated weapons require that aircrew achieve and maintain a performance capability at peak effectiveness. A vital aspect of this problem is the protection, preservation, and enhancement of aircrew capabilities through optimally effective physiologically based safety procedures, personal protective equipment, and other life support systems/subsystems.

This report documents the proceedings of an operationally oriented workshop that was intended to establish a comprehensive understanding of the current aircrew protective concepts, summarize lessons learned from earlier research, and uncover gaps in the knowledge base that might apply to protective requirements at altitudes of 60,000 feet and above. The thirty workshop presentations were grouped into six Sessions:, High Altitude Issues, Life Support Systems, Physiological Protection/Limits, Positive Pressure Breathing Effects, Current Protection Systems and Workshop Conclusions/Recommendations. A brief synopsis of individual papers is given for each session.

High Altitude Issues

The workshop session was opened with a review of the requirement for flight at altitudes of 60,000 feet and above. The need is driven by three primary factors: threat, weapons employment, and environmental conditions. With respect to threat, the combination of stealth, high speed, and high altitude operations will allow the aircraft to shrink some surface to air missile weapon engagement envelopes and totally neutralize others. As to weapons employment, the kinematic range of air to air missiles is greater at the higher altitudes. The major environmental factor requiring high altitude flight is to exploit stealth technology by climbing above contrails that, in summer months, can be seen at levels up to and including 60,000 feet. As engine performance continues to advance, there will be an ever-increasing drive to exploit the upper atmosphere and take advantage of the high ground.

Current USAF life support development efforts were reviewed. Presently, four user-supported and funded initiatives are in different stages of development: Combined Advanced Technology Enhanced Design G-Ensemble (COMBAT EDGE), Universal Water Activated Release System (UWARS), Night Vision System and Active Noise Reduction System. Other promising technologies awaiting user requirements, funding, and direction include: Advanced Technology Anti-G Suit (ATAGS); Combined Advanced Technology Acceleration/Chemical Ensemble (COMBAT ACE); Laser Eye Protection (LEP); and the ACES II ejection seat improvement program. Historically, the development approach has been to design each piece of equipment as part of an integrated whole, but in a modular fashion--wearing only those items required to accomplish the mission. A concept is being developed for the future that has its roots in the earlier Tactical Life Support System (TLSS), but there is currently no user-sponsored project involved with protection above 50,000 feet in the Life Support Program Office.

Physiological and operational factors were reviewed that determine the relationship between the concentration of oxygen in the inspired gas and cabin altitude in agile combat aircraft when the cabin is pressurized. The review considered in detail the disadvantages of breathing high concentrations of oxygen during aerial combat maneuvers. If acceleration-induced atelectasis and delayed otitic barotrauma are deemed to be unacceptable by aircrew operating agile combat aircraft, the standard that the maximum concentration of oxygen in the inspired gas shall not exceed 60% at cabin altitudes up to 15,000 feet (and 75% at cabin altitudes up to 20,000 feet), as required by Air Standard 61/22A and STANAG 3865, is sound.

Raising the ceiling of current flight operations will have the effect of increasing the altitude exposure hazard and consequent incidence of decompression sickness (DCS) symptoms. In some current operations, DCS is a limiting factor and may become the controlling element in mission planning for newer aircraft. For example, the F-22 pilot at a cruising altitude of 60,000 feet will have a cabin altitude of 22,500 feet, which is above the

threshold for DCS with a symptom onset latency of approximately one hour. Airdrop and reconnaissance missions are also impacted by the potential for DCS symptomatology. Prebreathing 100% oxygen or pure product gas from the molecular sieve prior to 16,000 feet exposure will provide significant protection against DCS. An approach to circumvent the disadvantages of acceleration-induced atelectasis and delayed otitic barotrauma associated with prebreathing higher concentrations of oxygen would be to increase the cabin pressurization differential from 5 psid to 6 or 7 psid, especially at higher altitude. DCS research should be directed at defining and predicting the risks and procedures for reducing DCS symptomatology.

A study has been approved at Farnborough to evaluate the risk of DCS and venous gas emboli (VGE) following loss of cabin pressure and subsequent sustained altitude exposure as required by current mission profiles. It is well understood that hypoxia can be prevented by standard breathing systems following descent to lower intermediate altitudes, but the risk of DCS is unknown. This is especially true following rapid decompression to altitudes ranging from 40,000 to 60,000 feet. Another protocol under development is to investigate the effect of rapid decompression on DCS incidence at higher altitudes, up to 60,000 feet. This program will support the Eurofighter 2000 program and is applicable to the F-22 program.

Flight at 60,000 feet with a 5 psid cabin pressure will expose the aircrew to 22,500 feet. Of concern is the potential for 1-2 hours of exposure to pressures above the threshold for DCS and subsequent rapid decompression to the lower ambient pressures at 60,000 feet. In this scenario, existing gas emboli will rapidly expand, resulting in potentially serious symptoms even during short excursions to these higher altitudes. This problem could be further exacerbated if descent to low altitude was not immediately possible. It is suggested that operational altitudes known to elicit high VGE counts in the majority of people should be avoided because of an increased risk of right-to-left gas cross-over to the arterial side of the circulation and the resulting potential of severe cerebral symptomatology.

Life Support Systems

The physiological and general requirements for high altitude breathing systems were reviewed, as well as some aspects of current design and compliance. The physiological factors include: adequate oxygen concentration at adequate pressure; provision of adequate nitrogen to avoid acceleration atelectasis; ventilation and flow levels; external resistance and removal of expirate. General requirements include: safety pressure; protection against toxic fumes and decompression sickness; evaluation of system integrity; indication of supply, flow and failure; simplicity of use; subsystem redundancy; protection during high-altitude escape and function in extreme environments.

Molecular Sieve Oxygen Concentrator (MSOC) technology has evolved into the dominant process for generating oxygen on-board military aircraft. A lucid review of the development of this technology was presented, with emphasis on USAF systems. Oxygen concentrator performance, operational use, backup oxygen capacity, and potential future technologies that would apply in oxygen concentrator systems were also reviewed. Future technologies include: the use of two zeolite molecular sieve beds and two carbon molecular sieve beds to produce up to 99.7% oxygen directly from compressed air; use of a zirconia ceramic membrane oxygen generating system to produce 99.9% oxygen; use of helium coldheads in conjunction with the MSOC to produce liquid oxygen; and, use of computer algorithms to automatically adjust concentrator operating parameters to accurately control product oxygen concentration while minimizing bleed air consumption.

The considerable interactions between the physiological requirements for cabin pressurization and the relationship between concentration of oxygen and cabin altitude required for aircrew oxygen delivery systems was reviewed. Both the F-22 and the Eurofighter 2000 use an isobaric 5 PSI differential pressurization schedule. With this schedule, normal cruise at 60,000 feet would yield a cabin altitude of 22,500 feet. The intensity of hypoxia acceptable following rapid decompression has important implications for the design of both pressure cabins and aircraft oxygen systems. Problems involving hypoxia as well as trapped gas issues are generally well established and most have been resolved with newer breathing systems. Recent research data, however, indicate that there would be a statistically significant risk of DCS with extended flight at cabin altitudes around 23,000 feet. The possibility of using a higher pressure differential at higher altitudes that would reduce the cabin altitude to around

18,000 feet or lower was discussed. It was noted that a further study of risk versus benefit would be required before a cogent position for changing pressurization schedules could be established.

Partial pressure systems designed for the F-22 and Eurofighter 2000 provide get-me-down protection from 60,000 feet. These systems are also designed to provide Nuclear, Biological, and Chemical (NBC) as well as Laser Eye protection. Using this fully enclosed head design as a starting point, it was suggested that it would not be a very large step to provide much longer protection to altitudes above 60,000 feet to reduce the immediate need for descent following rapid decompression. The changes would minimally include adding sleeves to the current vest, integrating the vest with uniform pressure trousers (ATAGS or equivalent), developing a pressure-containing head enclosure, mask and neck seal, redesigning the oxygen regulator/controller for manual as well as automatic operation of the pressure schedule, and providing pressure gloves. All of these concepts have been prototyped over the years. From lessons learned in these earlier studies coupled with newer materials and fabrication techniques, it was suggested that an improved, pilot-acceptable partial pressure helmet/suit system is well within the state-of-the-art. A methodical, iterative program should be developed to provide such an integrated high-altitude protective system.

Physiological Protection/Limits

Results of a study to determine the hypoxic risk of breathing 93% MSOC product gas versus 100% aviator's breathing oxygen to an altitude of 50,000 feet were reviewed. It was shown that the 93% or lower oxygen concentrations produced by the MSOC system provided significantly less protection than standard aviator's breathing oxygen. Conclusions include: oxygen monitors must provide reliable warning of MSOC product degradation; the current dilution schedule for standard regulators should be enriched at higher cabin altitudes to prevent transient hypoxia on rapid decompression; present positive pressure breathing schedules and mask sealing capabilities provide only marginal emergency descent protection at the current flight ceiling of 50,000 feet; and, a backup supply of 94% oxygen or greater is necessary to ensure post-decompression protection.

The principal physiological hazards associated with loss of cabin pressure at altitudes greater than 30,000 feet are hypoxia, DCS, and hypothermia. A full pressure suit assembly provides the ideal physiological protection but it is bulky, cumbersome, and impairs operational efficiency during flight as well as normal and emergency egress. A partial pressure assembly provides less restriction, lower thermal load, and generally greater routine comfort during flight. Thus, the design of partial pressure assemblies represents a compromise between ideal physiological requirements and functional convenience. The technique of pressure breathing with 100% oxygen and application of limited counterpressure to the body was adopted by the Royal Air Force in 1954 to provide short duration protection to altitudes as high as 60,000 feet. The main limitation in using an oronasal mask at pressures in excess of 70 mm Hg is discomfort resulting from the distension of the upper respiratory tract. Additional effects, such as blepharospasm (due to gas passing up the naso-lachrymal ducts) and rupture of the conjunctival vessels, limit breathing pressure and duration for which an oronasal mask may be used at 70 mm Hg to 3-4 minutes. It was suggested that mask pressures of 80 mm Hg accompanied by adequate body counter-pressure would probably be acceptable for short periods to 70,000 feet.

Over the past 15 years, a series of studies has been conducted at the Defense and Civil Institute of Environmental Medicine (DCIEM) in Canada to establish the high altitude limits of partial pressure assemblies. Following a lead from the Swedish Air Force, it was shown that protection against hypoxia was enhanced at higher altitudes with G-suit pressures in the range of 4 times mask-cavity pressures. The series of studies included exposure to: 56,000 feet for 3 minutes with 70 mm Hg positive pressure breathing (PPB); 60,000 feet for 3 minutes with 70 mm Hg PPB; 60,000 feet for 3 minutes with 60 mm Hg PPB; 72,000 feet for 2 minutes with 80 mm Hg PPB; and, finally, 80,000 feet for 1 minute with 80 mm Hg PPB. From these physiological and rather limited performance studies, it was postulated that emergency get-me-down escape was probably possible from altitudes as high as 80,000 feet. Subsequent flight trials to an altitude of 64,000 feet breathing 70 mm Hg with immediate descent to lower levels proved that a pilot could indeed control an aircraft following decompression to these higher altitudes. In subsequent PPB studies, it has been shown that subjects can tolerate up to 88 mm Hg for at least 20 minutes with full coverage lower body G-suits pressurized to 4 times breathing pressures.

Positive Pressure Breathing

Results of an invasive cardiopulmonary study were reviewed that investigated the cause of the hyperventilation associated with PPB using a COMBAT EDGE assembly. Results indicated: a 5-fold increase in minute ventilation with 60 mm Hg PPB; the increase in minute ventilation was achieved by an increase in tidal volume and not frequency of breathing; the higher mask pressure resulted in only a slight reduction in cardiac output; phasic swings in mask pressure seem to augment venous return and sustain arterial pressure; increased G-suit pressure may augment blood return and maintain arterial pressure but have unknown effects on overall pulmonary gas exchange; heart rate was higher and arterial pressure was lower at altitude than ground level controls; and, a discrepancy was shown between end tidal and arterial partial pressure of carbon dioxide ($PCO2$) while pressure breathing, which indicated to these authors that assessment of arterial $PCO2$ during PPB should ideally be obtained by direct measurement of arterial gas tension.

The reality of routine flight at 60,000 feet and above will demand that aircrew are protected not only for get-me-down protection as in the past, but possibly even mission completion scenarios after decompression. Results from a series of studies at the DCIEM using full-coverage anti-G trousers at various gradations of inflation ratios and 60 mm Hg PPB indicate that a 3-to-1 pressure ratio between the vest and trousers caused a slowing of heart rate and a 2 to 1 ratio provided equal cardiovascular protection. It was concluded that the new full-coverage G-suit affords vastly superior bladder coverage over the limbs, optimized pressure transmission to the tissues, and consequently do not require the traditional 4-to-1 pressure schedule originally developed for use with the older CSU-13/BP G-suit. The lower ratio still affords adequate left ventricular filling and stroke volume, while allowing a more appropriate heart rate response. Lower inflation pressure without the loss of physiological function also addresses the problem of comfort during extended duration PPB.

Although there have been no documented medical complications in the pressure range that has been established for aircrew breathing systems, it is important to be aware of several potential medical conditions during the development of new PPB schedules. These conditions would include: pneumothorax, pneumomediastinum, surgical emphysema, arterial gas embolism, hypertension, hearing damage, and problems associated with raised intraocular pressure (IOP). A study has been recently initiated to investigate the effects of IOP during exposure to PPB levels up to 60 mm Hg for periods up to 10 minutes, and to determine the fall in IOP pressure following cessation of PPB. The observations to date indicate that IOP does increase with PPB, although not on a one-to-one basis and, upon cessation of PPB, the IOP usually returns to baseline levels within 2-6 minutes.

At the present time, no comprehensive model can describe the physiologic consequences of a change in operating conditions and design of the life support systems employed in modern high-performance aircraft. Several tentative computer models of components required to implement an integrated model of the Aviators Breathing System (ABS) have been created. A fully validated model could be used to simulate the function of the hardware and the physiological effects in response to environmental changes. The fully integrated ABS model could be employed to perform parametric studies as an aid to illustrating and understanding the nature of the interactions between the environment, the life support system and the aviator. Ideally, the final model would be a single software module with a user-friendly interface that would allow easy selection and adjustment of input variables and display of simulation results.

Current lung overpressure schedules are based on poorly understood human tolerance limits. The safe limits for static pressure are conservatively set at 60-100 mm Hg and for supported static pressures at 170-190 mm Hg. These limits do not account for dynamic overpressure situations. Using current human data, the unsupported aviator should be able to tolerate: the standard 5 psi (259 mm Hg) overpressure occurring in 0.06 seconds or greater; 6 psi (310 mm Hg) overpressure in 0.08 seconds or greater; and a 7 psi (362 mm Hg) overpressure in 0.1 seconds or greater. Animal evidence suggests that even greater pressure could be tolerated in humans with minimal harmful effects. Use of well-designed flight gear, providing chest and abdominal counter-pressure, increases human pulmonary overpressure tolerance primarily through increased thorax rigidity. Use of well-designed flight gear should increase the safety margin for tolerance to a 7 psi decompression in 0.01 seconds in duration.

Positive pressure breathing increases altitude tolerance by producing an increase in inspired, and hence alveolar, PO_2. The magnitude of this increase is most closely related to mean airway pressure. This airway pressure elevation has effects that can reduce oxygen delivery and result in significant pilot morbidity, e.g., reduced cardiac output, hyperventilation, and pulmonary barotrauma. Various alternatives to the standard pressure breathing profiles were reviewed. These alternative methods included: augmenting the area of coverage of the protective suit, i.e., the more closely a partial-coverage suit mimics a full-pressure suit, the less physiological derangement will occur; phasically increase pressure during inspiration (analogous to pressure support ventilation) or expiration (analogous to expiratory positive airway pressure) to augment venous return. Hence, cardiac output and pulsing the respiratory system at high frequency may be useful by attenuating PPB-induced hyperventilation and pulsing the airway during late diastole to augment cardiac output. Replacement of continuous PPB with a system providing phasic pressure alterations has the potential to augment cardiac output and tissue oxygen delivery, while minimizing hyperventilation.

In recent studies at Farnborough, it was shown that a counterpressure assembly consisting of waistcoat and anti-G trousers, when inflated uniformly to breathing pressure (1-to-1 ratio) provided less support to the cardiovascular system that the standard RAF counterpressure assembly. Inflation of the lower counterpressure garment to three times breathing pressure (3-to-1 ratio) caused highly significant improvements in the physiological protection. The arterial pressure elevated closer to the sum of the resting blood pressure and PPB, and a more satisfactory pulse pressure was observed, as well as a reduction of the tachycardia associated with PPB. Increasing the lower garment inflation to four times breathing pressure did not further improve the protection provided.

With the use of chest counterpressure, extended coverage anti-G suits, and a 4-to-1 G-suit-to-PPB pressure ratio, it has recently been demonstrated that well-trained subjects can tolerate breathing pressures of 60 to 70 mm Hg for well over 10 minutes, and as much as 20 minutes. Indeed, four of six subjects tolerated 80 mm Hg for 20 minutes. It was also suggested that there may be considerable utility in using PPB to increase absolute body pressure and thus reduce the risk of DCS following loss of cabin pressure at higher altitudes. For example, if an individual were exposed to 45,000 ft breathing oxygen at either 30 mm Hg or 80 mm Hg, the effective physiological altitude considering DCS symptomatology would be around 40,000 ft for the former and below 34,000 ft for the latter. This possibility remains to be proven, but is an interesting speculation.

Current Protection Systems

The Tactical Life Support System (TLSS) was the first USAF advanced development program. It was a highly ambitious effort that guided design requirements to provide high-altitude protection, improved G protection, personal cooling, and improved NBC protection. As an advanced development program, TLSS was intended to provide a vehicle to incorporate the laboratory-generated technical advancements into an integrated system to improve aircrew life support. This mid-1980s effort spawned most of the current improvements in life-support equipment now being prototyped for the F-22 aircraft, as well as the current COMBAT EDGE system being flown in the F-16 aircraft and shortly to be deployed in the F-15 aircraft.

Positive pressure breathing, in combination with counterpressure to the thorax by a vest bladder, has been introduced to increase the endurance tolerance of high G-loads. Such a system, i.e., the COMBAT EDGE, is now in operational use in the USAF F-16. This system gives a linear increase of the breathing pressure from +4 Gz to a maximum of 60 mm Hg at +9 Gz with the same pressure in the thoracic vest bladder. The Swedish use a breathing pressure increasing from 3 mm Hg at +4 Gz linearly to about 50 mm Hg at +9Gz, while the French are currently testing a maximum breathing pressure of about 70 mm Hg. Unassisted PPB has been shown to reduce or eliminate G-induced pulmonary atelectasis during oxygen breathing; however, the measure of atelectasis while breathing greater than 70% oxygen with assisted PPB during increased G-loads has not yet been evaluated.

The F-22 life support program did not set out to create a system to expand the fighter communities altitude environment, but rather to use the inherent altitude protection characteristics afforded by the partial-pressure garments for G protection. The system offers protection for inflight decompression and post ejection to

altitudes in excess of 50,000 feet. The complete partial pressure assembly, in combination with the Molecular Sieve Oxygen Concentrator (MSOC), is viewed as only get-me-down protection. The F-22 life-support system is composed of both aircraft and man-mounted hardware. Aircraft equipment is comprised of the three-bed MSOC, Breathing Regulator and Anti-G (BRAG) valve, and seat-mounted Emergency Oxygen System. Man-mounted equipment is comprised of the integrated terminal block, upper pressure garment, air cooling garment, lower G garment, helmet, and mask.

The Eurofighter life support system will incorporate state-of-the-art anti-G breathing systems with Aircrew Equipment Assemblies comprising full-coverage anti-G trousers, chest counter-pressure garment and pressure demand oronasal mask with enhanced mask sealing. Some of the physiological challenges involving acceleration protection that remain to be established include: optimization of the PPB schedule; G threshold for onset of pressure, slope of the PPB schedule and maximum PPB; minimizing arm pain from the PPB; and, effect of loss of cabin pressure during PPB. Regarding altitude protection, the following remain to be established: the pressure ratio between anti-G trousers and mask; MSOC gas composition prior to decompression; confirmation of the acceptability of 80 mm Hg PPB; and, effects of trapped gas in the NBC assembly.

Following a review of full-pressure suit operations for reconnaissance aircraft, it was concluded that today's high-performance flight operations would be significantly degraded if a current full-pressure system were required. It was noted that there are numerous physiological, psychological, physical, and performance limitations associated with wearing a full-pressure suit. The physiological support infrastructure required to maintain the operational readiness of the U-2 fleet is robust. Expanding this responsibility to a larger, more diverse complement of weapon systems cannot be accomplished with current resources. To do so would require designing an innovative suit capable of self-donning and doffing, with integrated self-diagnostics and disposable suit components, and reduced bulk, especially over the extremities. The final suit configuration must be capable of flawless, sustained performance without the current level of required resources and logistic support.

Many variables must be considered when attempting to circumscribe the acceptable risks of sustained operations at high altitude. Some of the factors for remaining at high altitude following loss of cabin pressure include: flying over a surface-to-air missile belt; improving fuel consumption; better reconnaissance (over the horizon); greater kinetic energy of the missile; and a higher speed making it more difficult for a gunner to target the aircraft. Although a risk analysis and benefit study was indicated, it was suggested that a well-trained aircrew member, wearing the high-pressure mask, vest, and uniform-pressure anti-G assembly contemplated for the F-22 or Eurofighter 2000, should be able to tolerate the hypoxia associated with exposure to 65,000 feet for 5 minutes, providing he could immediately descend to lower altitudes. The immediate risks involved the ability of the aircrew to breathe at pressures around 75 mm Hg without hyperventilating, and the capability of the MSOC or backup oxygen to provide adequate pressure/flow/concentration of oxygen. Questions remained whether DCS would become a major risk at this altitude for the 5-minute period. This depends in great degree on whether the aircrew was exposed to a cabin altitude of 23,000 feet or greater for a long period before decompression, and whether there was an adequate denitrogenation period.

The workshop provided the opportunity for a thorough review of the need for flight operations at 60,000 feet and above, as well as an open scientific discussion of the physiological consequences and exposure limits at these altitudes. A summarized listing of the most important conclusions and recommendations coming out of the workshop is on the following page.

RAISING THE OPERATIONAL CEILING - A WORKSHOP ON THE LIFE SUPPORT AND PHYSIOLOGICAL ISSUES OF FLIGHT AT 60,000 FEET AND ABOVE.

BROOKS AFB, TEXAS 13-15 JUNE 1995

Conclusions/Recommendations

- A definite need for operational flight at 60,000 feet and above was established. Flight at these altitudes provides several important combat advantages.

- An urgent requirement exists to determine the operational impact of breathing intermediate concentrations of oxygen to reduce G-induced atelectasis at lower cabin altitudes versus breathing higher concentrations oxygen to decrease the risk of hypoxia and decompression sickness following loss of cabin pressure.

- There is a magnified risk of DCS following loss of pressure at high altitude and descent to intermediate unpressurized altitudes on return to home base, especially if the aircrew were predisposed by exposure to high cabin altitudes before the decompression.

- Although it has been demonstrated in laboratory studies that individuals can tolerate high levels of PPB using the current concept, mask/vest/full-coverage anti-G suit assembly with appropriate pressure ratio, it was concluded that we are nearing the edge of human tolerance limits with operational exposure at altitudes of 60,000 feet and above using these assemblies. Further research on the cardiopulmonary effects of PPB and alternative methods of maintaining cerebral oxygen tension were indicated.

- If it becomes necessary to fly above 65,000 feet, a likely approach for tactical aircraft would be to use a fully enclosed helmet containing a high-pressure mask, sleeved vest and ATAGS-equivalent partial-pressure assembly. For many reasons, a full-pressure suit assembly is not considered a practical alternative.

- An advanced hybrid oxygen system (AHOS), which provides nearly 100% oxygen from the MSOC product in either gas or liquid form, holds great promise for the future to reduce the risk of hypoxia at higher altitude.

- Using operational flight scenarios, studies should immediately be directed toward definition and prediction of the risks of DCS to conclusively document thresholds and onset times.

- Increasing the aircraft environmental control system schedule above the standard 5 PSID isobaric differential should be investigated as a means of reducing exposure to the high cabin altitude in higher flying aircraft.

- The emergency oxygen system in future F-22 derivatives should be optimized by: increasing the volume of the system to at least 200 liters; providing a manual on/off function, as well as automatic operation; and, providing sensors to indicate the status of the oxygen system.

- Pressure breathing instruction and practice, related to high-altitude flight, must be included in the fighter training syllabus.

- An interactive computerized database is needed to ensure that the interrelationships between aircraft/crew/physiological issues are considered in the design of future aircraft and life-support systems.
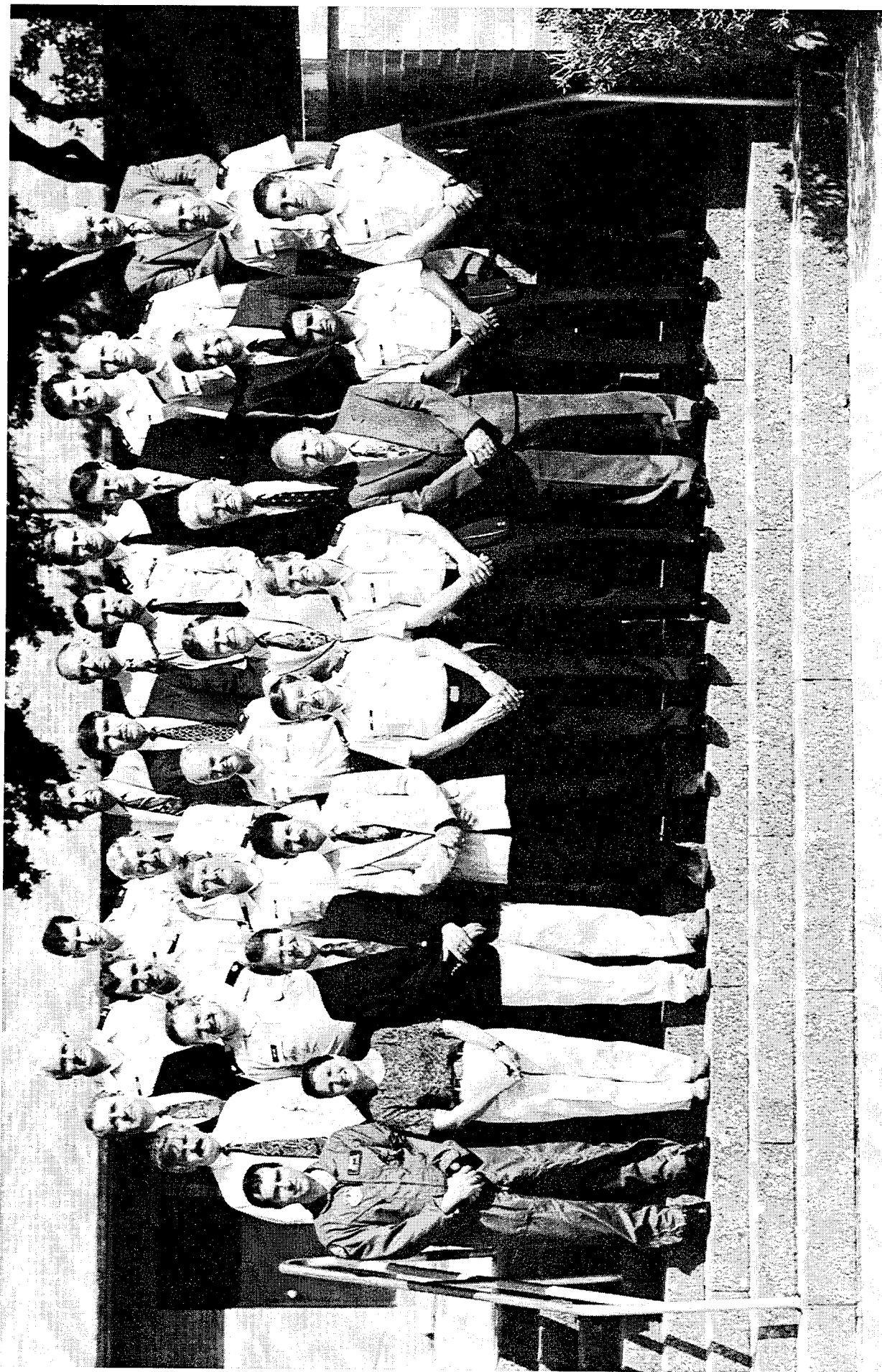
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RAISING THE OPERATIONAL CEILING--
WORKSHOP ON THE LIFE SUPPORT AND PHYSIOLOGICAL ISSUES
OF FLIGHT AT 60,000 FEET AND ABOVE

JUNE 13-15, 1995

BROOKS AIR FORCE BASE, TEXAS

CREW TECHNOLOGY DIVISION WELCOME

Colonel James Dixon
Chief, Crew Technology Division
Brooks Air Force Base, Texas 78235-5301

I want to thank each of you for being present at this workshop on the physiological issues of raising the operational ceiling to 60,000 feet and above. On behalf of the Director of the Armstrong Laboratory, Dr. Brendan Godfrey, and our new commander at the Armstrong Laboratory, Colonel Terry Lyons, I want to extend a heartfelt welcome. Colonel Lyons could not be here with us this morning, but he sends his encouragement for a productive workshop.

Both Colonel Lyons and I are very pleased with the notable qualifications and talent of the participants at this workshop. We have high expectations that we can use the guidance generated at this meeting to provide protective measures and other innovative concepts to allow the aircrew to fly at higher altitudes.

Until quite recently, flight operations at high altitude had lost some of the interest it formerly held. The extended capabilities of newer high-performance aircraft, as well as newer air- and ground-based threats, now make it necessary for the scientific community to extend the capabilities of the human--in this case, to fly at altitudes previously reserved for those wearing full-pressure suits. Dr. Andy Pilmanis and his colleagues have recently demonstrated the risks and potential limitations of exposing humans for longer periods of time to the higher cabin altitudes associated with high-altitude flight. We are in the process of redirecting our energies to ensure that flight at these higher altitudes will not degrade the performance of the aircrew, while providing them with protection against all contingencies. We are expecting that this gathering of international expertise will provide us with many of the answers and alternatives to the varied problems associated with high-altitude flight in advanced technology fighter aircraft.

As each of you interact, and perhaps generate action items for us in the Armstrong Laboratory, we fully anticipate the products of this conference will provide a lasting positive influence on our various operational forces. Again, on behalf of Dr. Godfrey and Col. Lyons, we welcome you and wish you the very best in your efforts here this week.

SCOPE, ISSUES, AND OBJECTIVES OF THE WORKSHOP

Andrew A. Pilmanis Ph.D.
William J. Sears Col USAF (Ret) Ph.D.

The altitude ceiling for future fighter aircraft such as the F-22 and Eurofighter 2000 has been raised from 50,000 to 60,000 feet. This 10,000 feet increase greatly increases the physiological hazards for the crew as well as the life-support system requirements (Table 1). The impact of this change is complex and could impose decisive limitations with regard to crew safety issues. A comprehensive and integrated approach is necessary to ensure that crew safety is maintained. Clear definition of both physiological and systems limitations is required in order to develop trade-offs between operational requirements and crew effectiveness.

Table 1. Complexity of issues involved in raising the ceiling.

AIRCRAFT/CREW ISSUES	PHYSIOLOGICAL ISSUES	LIFE SUPPORT SYSTEMS	
FLIGHT SCENARIOS	**HYPOXIA**	**OXYGEN SYSTEMS**	
FLIGHT ALTITUDE	OXYGEN CONCENTRATION REQUIRED	MSOC	VOLUME/FLOW REQUIREMENTS
DURATION OF FLIGHT	OXYGEN PRESSURE/FLOW SCHEDULES		CONCENTRATION SCHEDULES
	TIME OF EXPOSURE TO ALTITUDE		FILTERED AIR BYPASS
CABIN PRESSURIZATION	BREATHING RESISTANCE		PURGE VALVES
	BREATHING PRESSURE SWINGS		SENSORS/INDICATORS
CABIN PRESSURE DIFFERENTIAL			
CABIN VOLUME	**DECOMPRESSION SICKNESS**	LOX	CONVERTERS
SIZE ORIFICE			SUPPLY REQUIREMENTS
DECOMPRESSION RATE	BENDS, CHOKES, CNS DISTURBANCES		STORAGE REQUIREMENTS
TRANSIENT CABIN PRESSURE AFTER RD	EXPOSURE TIMES		INDICATORS/REGULATORS
FLIGHT ALTITUDE REQUIRED POST RD	PREBREATHE REQUIREMENTS		GAUGES/HEAT EXCHANGERS
	WORKLOAD		
HUMAN FACTORS		GAS	HIGH PRESSURES
	POSITIVE PRESSURE BREATHING		SUPPLY REQUIREMENTS
PERFORMANCE DEGRADATION			VOLUME/FLOW REQUIREMENTS
WORKSPACE LIMITATIONS	PRESSURE/FLOW REQUIREMENTS		BACKUP TO MSOC
EQUIPMENT ACCEPTABILITY	CARDIOVASCULAR EFFECTS		EMERGENCY OXYGEN
FIT/FUNCTION OF EQUIPMENT	RELATIVE GAS EXPANSION		
CREW COMFORT	HYPERVENTILATION		
CREW SAFETY	PRESSURE BREATHING LIMITS	**REGULATORS**	
	MASK VERSUS INTRATHORACIC PRESSURES		CONCENTRATION SCHEDULES
	PULMONARY OVERPRESSURE		PRESSURE SCHEDULES
	GAS EMBOLISM		DILUTER DEMAND VS 100%
	PNEUMOTHORAX		DELIVERY RATES
	TRAINING		INLET/OUTLET PRESSURES
			BREATHING RESISTANCE
	EBULLISM		OSCILLATORY BEHAVIOR
			RELIEF VALVES
	ALTITUDES/DURATION OF EXPOSURE		PANEL /SEAT/MAN MOUNTING
	UNPRESSURIZED AREAS OF BODY		VEST/NO VEST PRESS SCHEDULE
	SHORT/LONG TERM EFFECTS		INDICATORS/CONNECTORS
			SHUT OFF VALVES
	TRAPPED GAS		
		MASKS	RETENTION CAPABILITIES
	PULMONARY OVERPRESSURE		AUTO/MANUAL TENSIONING
	RAPID DECOMPRESSION		MASK CAVITY PRESSURES
	DELAYED EAR BLOCK		BREATHING RESISTANCE
	G-INDUCED ATELECTASIS		PRESSURE COMPENSATION
	SINUSES		QUICK DISCONNECT WARNING
	GI TRACT		COMFORT
	THERMAL	**PRESSURE ENSEMBLES**	
	TEMPERATURE	MASK/VEST/G-SUIT	
	DURATION OF EXPOSURE		PBA SCHEDULES
	PROTECTIVE CLOTHING		PBG SCHEDULES
			POST EJECTION SCHEDULES
			DUAL ANTI-G SUIT BLADDERS
			SLEEVED VEST/VENOUS POOLING
			FULLY ENCLOSED MASK/HELMET
			ISOLATION VALVES
			MASK/VEST DIFFERENTIALS
			MAX ACCEPTABLE PROTECTION
		PARTIAL-PRESSURE SUIT/ENCLOSED HELMET	
		PRESSURE SCHEDULE 140 TORR ABS	
		FULL-PRESSURE SUIT/ENCLOSED HELMET	
		PRESSURE SCHEDULE 180 TORR ABS	

Much of the rich scientific database on exposure to high altitude has been reported at annual meetings and in journals. As a result of publication delays and limited attendance at the meetings, these reporting methods are often inadequate for the dissemination of information. Furthermore, individuals may be expert and focused in one topic area while relatively uninformed in areas interrelated with the specific topic field. One of the primary objectives of this workshop is to more clearly define and update these interrelationships. This is a complex challenge. It is anticipated that, following the workshop, a computerized matrix containing the interrelationships between the items listed in Table 1 will be formulated and used in the future development of high-altitude protective ensembles and safety procedures.

Specific Workshop Objectives

- To outline the rationale for operating at higher altitudes.

- To define current life-support equipment capabilities.

- To review and integrate the physiological and life-support requirements for increasing the operational altitude.

- To establish physiological and safety limits.

- To initiate development of an interrelated life-support matrix.

- To identify operational research requirements.

- To provide a timely publication covering relevant issues to prevent crew safety from becoming a limiting factor in future high-altitude operations.

Sample Discussion Questions for the Workshop

- What are the maximum +Gz limits that can be expected in high-performance aircraft at altitudes between 40,000 and 65,000 ft? What +Gz level causes operationally significant G-induced atelectasis?

- What is the highest concentration of oxygen that can be used in high-performance aircraft as a compromise between the safety aspects of +Gz induced atelectasis and the potential for hypoxia/decompression sickness post RD?

- What is the breathing gas composition in the lung during PBG at +9Gz in most of the newer mask/vest/anti-G pressure systems?

- What is the optimum size of the emergency oxygen system for ejection, as well as for smoke and suspected contamination during stand-by and flight?

- What are the actual free-fall times in an ejection seat from 60,000 feet? During ejection, do most of the newer get-me-down pressure ensembles pressurize the trousers higher that the mask/vest?

- Is it necessary to install a 100% backup oxygen system in case of rapid decompression or is the pressurized MSOC product gas adequate for short-term exposure to 60,000 feet? 65,000 feet?

- What is the operational high-altitude limit for the mask/vest/anti-G suit ensemble considering the hypoxia and DCS risks and remaining flight time to base? To what lower flight altitude should the aircrew be exposed for the return flight? Does this limit take into consideration the potentially fatigued condition of the aircrew prior to exposure to high altitude?

- What is the feasibility of using a higher cabin pressure differential, e.g., 6-7 PSI, to reduce the DCS risk associated with higher cabin altitudes and rapid decompression? What about the possible use of a two-pressure cabin pressure controller, depending on whether the crewmembers were wearing the mask/vest/anti-G ensemble versus a mask alone?

- What are the maximum safe intrapulmonary pressures for pressure breathing with and without chest counterpressure? Sustained and peak pressure breathing?

- Are there significant ventilation/perfusion problems associated with PBA at high altitude?

- What are the optimum regulator PBA schedules for the mask/vest/anti-G pressure ensemble? In case the vest is not worn?

- What are the maximum safe times for sustained pressure breathing between 30 to 80 torr?

- What are the DCS risks and onset times at altitudes above 40,000 feet? After being exposed for long periods at cabin pressures above 20,000 feet? After return to lower altitudes following RD?

- Should sleeved vests and full head helmets be required at altitudes above 65,000 feet? Can anyone envision a comfortable and crew acceptable full-pressure suit?

- Is there a mission scenario that would require continued flight above 50,000 feet following decompression - even for periods up to 10 minutes?

- Does anyone anticipate a problem with ebullism with short exposures to altitudes above 60,000 feet?

- Have PBA schedules in the get-me-down partial-pressure ensembles taken into account the aerodynamic reduction in the cockpit pressure following rupture or loss of the canopy?

- What are the training requirements for PBA? What training devices are available?

Discussion

COL. DIXON: Dr. Pilmanis, would you encourage the participants to make a prepared statement of certain issues that they feel are most important to them?

DR. PILMANIS: Absolutely. We would also encourage those who present a specific topic area to make recommendations and conclusions, and if there's consensus we can enter it directly into the proceedings.

F-22 Concept of Operations
Above 50,000-ft

Greg Neubeck, Major, USAF

Introduction

With the introduction of three new Soviet fighters in the 1980s, the Soviets have realized unprecedented gains in tactical air power. In the early years of the 21st century, MIG-29, -31 and Su-27 fighters will be joined by two potent newcomers, an air superiority fighter and a counter-air fighter. These new fighters are expected to incorporate low observables, improved cockpit technology, striking improvements in maneuverability, and the capability to sustain operations above 50,000 ft. Like their predecessors, these fighters will be able to fight effectively at night and during bad weather. With advanced sensor systems, they will possess an all-important first-look, first-shot capability against current fighters.

While the overall posture and actual numbers of the Soviet defense force may be slightly altered, we can expect to see continued investment in research and development, as well as the fielding of new weapon systems in the 21st century. Even if the Soviets make substantial reductions in their military expenditures, it is important to remember that these cuts would begin from extremely high levels of force structure and production rates.

Clearly, there is a mandate for a viable air superiority capability. To excel in the perilous air combat arena of the future, the US must fly a tactical fighter with unmatched abilities. That innovative aircraft is the Advanced Tactical Fighter - ATF. This paper will address the F-22 balanced design concepts and why flight above 50,000 ft is feasible and also necessary.

A Balanced Design

The F-22 project has incorporated a balanced design to foster affordability; enhance survivability, increased reliability and maintainability; and stresses performance to achieve air superiority for the 21st century. Upgrading current fighters would not be a more affordable solution than procuring the ATF. In fact, the ATF will cost no more to own and operate than current air superiority fighters, and it will give Air Force and Navy tactical forces vastly improved combat effectiveness. Current fighters, even with improvements, cannot approach the combat effectiveness of the ATF. The process of upgrading F-14s, F-15s and F-16s with new engines, avionics and weapons would be exceedingly costly. And one must not forget that the "modified" aircraft would require, among other things, more replacement parts and higher logistics costs than the ATF. In fact, the long-term impact on the budget for maintaining air superiority with the ATF is substantially less than upgrading today's aircraft. An affordable design also means sustaining operations in a high-threat environment by producing substantial kill ratios in favor of the ATF. To accomplish this goal there are five areas critical to the design: stealth, acceleration, supercruise, maneuverability, and radar detection range.

The ATF employs a mixture of revolutionary technologies that will enhance its survivability and performance. The ATF's stealthy attributes reduce significantly the range at which it can be detected. The fighter's contoured shapes diminish its radar cross section, while composite materials, special skins, and coatings absorb or deflect radar energy. Thermal signature is also markedly reduced to decrease the likelihood of detection by infrared sensors. The ATF's stealth characteristics dramatically confound the enemy's situational awareness and increase the probability of tactical surprise. Success in aerial combat will be determined by whoever obtains the tactical advantage--first look. Reduction in signature results in substantial gains in offensive potential and survivability.

At the heart of the ATF is the most sophisticated avionics architecture ever engineered for air-to-air (A/A) combat. The aircraft's electronic sensors detect, identify and react to multiple threats. In a combat environment, the ATF's integrated avionics will enable the pilot to avoid task saturation, freeing him to concentrate on the fight at hand. Fully integrated avionics consolidate threat-warning and countermeasures, so defensive and offensive systems work in unison. Stealth and supercruise set the ATF apart from any of today's air superiority fighters, East or West. The ATF's unparalleled combat effectiveness arises from its ability to elude enemy radars while detecting A/A threats at ranges that will provide the F-22 pilot a critical tactical advantage with first-look, first-shot capability. With the sophisticated air-to-air missiles carried in the ATF's internal weapons bay, that first shot will inevitably translate into a first kill.

With its supercruise capability, the ATF has superior acceleration and sustained energy in the transonic region. Rapid acceleration to combat speeds will allow the F-22 to effectively separate from within visual range (WVR) threat weapons envelopes. Acceleration, coupled with the ATF's ability to vector thrust, will enable the pilot to control the initial setup of the fight, maintain the offensive, and accomplish a quick kill. During air combat maneuvers, the ATF will rely on its agility and nose-pointing authority to defeat the enemy. These are critical attributes that could mean the difference between survival and failure.

The ATF will possess the ability to fly at supersonic speeds without the need for afterburners, which means the ability to sustain high-speed flight without the same high fuel consumption required by conventional fighters. The ability to sustain high speeds will allow the F-22 to minimize exposure time to surface-to-air missiles (SAMs).

The F-22, using its advanced airframe design, control surfaces, vectored-thrust engines, supercruise capability, and integrated avionics, will maintain the edge in close-in combat. The aircraft can kill from long range, but if it enters into close-in combat, the F-22's superior agility will provide US pilots with an important advantage. The ATF will take the fight to the enemy at any time, day or night, low and high altitude, and in all weather.

Why Go High

The ability to achieve the high ground has been an influence on fighter aircraft design since the beginning of aerial combat. In World War I fighter aircraft flew to higher altitudes to allow for high-speed attack and separation on an opponent at a lower altitude. Aircraft altitude limits during this time were due to aircraft performance and the maximum altitude was 22,900 ft. By the time WW II began, aircraft were operating above 40,000 ft. For the first time, aircrews required life-support systems to allow operations at these altitudes for extended periods of time. Once again, aircraft were pushing the combat ceiling higher and higher attempting to achieve the advantage of the high ground.

The 1960s introduced the SR-71, the first aircraft to take off and land like normal aircraft but operate at altitudes of 70,000 ft plus. The SR-71, dedicated to reconnaissance gathering over or near enemy territory, used high altitude and high speeds to elude enemy aircraft and SAMs. Today's conventional aircraft are limited to operations below 50,000 ft due to limitations of life-support systems and aircraft engine performance. The MIG-25, -31, F-22 and the new Eurofighter 2000 are now pushing the operating envelope beyond 50,000 ft, once again competing for the high ground.

The F-22 will operate above 50,000 ft due to superior engine performance. The F-22 incorporates a positive pressure breathing (PPB) system and full-coverage lower- and upper-torso G garments to provide the pilot added G protection. These systems will also allow the pilot to operate above 50,000 ft for extended periods of time under normal conditions and for brief times under cockpit depressurizations. The F-22 life-support system is designed as a get-me-down system in a cockpit depressurization. It will allow the pilot to get down to safer altitudes and reduce the chances experiencing decompression sickness.

The reasons to go high are driven by three areas: threat, weapons employment, and environment. The threat is comprised of surface and air threats. The surface threat, comprised of SAMs and AAA, will impede the progress of any conventional force. Eliminating or denying surface threats a chance to engage is key to survival when crossing the forward edge of the battle area (FEBA). Conventional aircraft crossing the FEBA will have a difficult time getting through; their survival will depend upon the use of their own self-defense equipment, as well as relying on the help of airborne jamming assets and aircraft with SAM suppression weapons. The combination of stealth, high-speed, and high-altitude operations will allow the F-22 to shrink some SAM weapon engagement envelopes (WEZ) and totally neutralize others. This will also allow the F-22 to operate independently of support assets thereby making employment more flexible.

The kinematic range of A/A missiles improves at the higher altitudes. Higher altitudes impose less drag on missiles allowing them to maintain higher energy states and therefore increase their maximum kinematic range. In contrast, a missile shot from a lower altitude at a higher altitude target must first climb through the denser atmosphere imposing a higher drag on the missile. The combination of increased drag and the requirement of the missile to climb to intercept the target reduces its energy early in the intercept and consequently reduces its maximum kinematic range. The difference in launch ranges between high-to-low and low-to-high shots can be as much as 6-10%. Therefore, aircraft will strive to get higher than their adversary to achieve a kinematic advantage for their A/A missiles, which translates into achieving the first-shot and possibly the first-kill.

Nothing is absolutely invisible. The whole idea of low observables is to balance the design that factors in all the detectable signatures. This paper has already discussed the benefits of the F-22's stealth characteristics but stealth also includes suppressing acoustic, electromagnetic, visual, infrared, and environmental effects. The environment confronts pilots with many variables forcing them to make changes to their tactical game plan simply based on the weather. The environment is incorporated in the tactical planning for every mission that is flown regardless of whether it is training or combat. On a clear day an aircraft leaving a contrail behind can be seen for hundreds of miles undermining the benefits of stealth. To take advantage of the high ground and maintain the advantages of stealth aircraft, will have to climb above the contrails (cons). Figure 1 shows the average minimum and maximum con levels for a particular area of the world. As can be seen, the highest con level occurs in the summer months (up to and including 60,000 ft) and decreases during the winter months. Conventional aircraft stuck below 50,000 ft will spend much of their time in the cons when operating at 40,000 ft and higher. This single factor is very important to the overall tactical situation. The tactical situation dictates being undetected as the key to success and therefore the cons should be avoided. If con penetration it required, it must be done quickly or far enough away to preclude an enemy from witnessing the transition. There have been more A/A kills as a direct result of a pilot gaining a tally (visual sighting) on his adversary than through the help of any other sensor developed (i.e., radar). As much as we have spent on stealth technology, it could all be for naught if one F-22 was caught in the cons and shot down.

There are disadvantages in going high: aircraft performance and Infrared (IR) sensors. In order to achieve high-altitude flight above 50,000 ft, aircraft must use afterburner. Afterburner operations will use fuel at a faster rate, consequently reducing flight duration time. Also, aircraft pay a great expense if aggressive maneuvering is required to react to a threat. As an example, an aircraft executing a 4-g turn at 50,000 ft will lose 12,000 ft of altitude. Aircraft operating at the higher altitudes must maneuver conservatively in order to remain there.

In a standard atmosphere, temperature and moisture decrease with increasing altitude. This effect lessens infrared (IR) attenuation, improving the detection capability of an IR sensor. IR sensors can detect objects at greater distances in the higher altitudes. Today, many foreign aircraft are using Infrared Search and Track Sets (IRSTS) to detect aircraft by the heat source generated by their engines. As mentioned earlier, it takes afterburner to achieve and sustain operations above 50,000 ft, which makes a stealth aircraft more vulnerable to detection by an IR sensor. Designers continue to work to suppress the heat signature of an aircraft by diffusing or mixing the heat source with the cooler outside air. However, IR signatures cannot be completely eliminated and the capability to do so will continue to challenge designers for future aircraft.

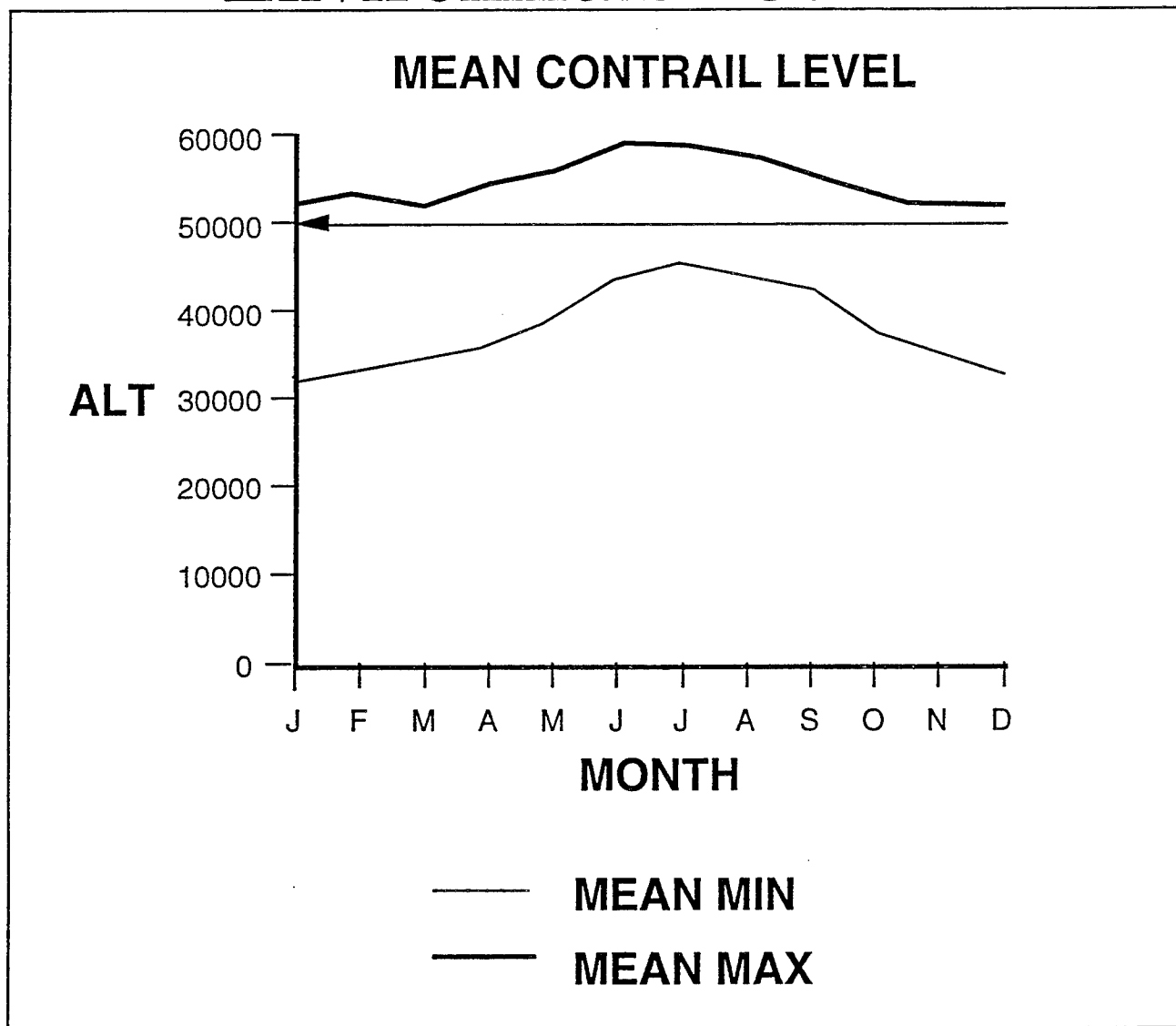# Advantages of Going High Environment - Contrails

Figure 1. Contrail Level.

Operating at Altitude

The SR-71 and U-2 placed early demands on life support systems to accommodate aircrews who flew at 70,000 ft plus. To sustain life in the hostile environment of the upper atmosphere, the only alternative for the pilot was to use a pressure suit. However, in the tactical environment of A/A combat, today's pressure suits would not be the answer. The life-support ensemble required to support the fighter pilot must be lightweight; provide good mobility within the cockpit; protect the pilot under high G; not impair the pilot's visibility; reduce heat stress; protect against the chemical and biological threat; and allow the pilot to operate without impairment throughout the limits of the aircraft, including sustained operations above 50,000 ft. Today's life-support ensemble does not come close to meeting all these requirements. A life-support ensemble is needed that integrates all these requirements into one package. Today's technology has taken giant strides in providing highly maneuverable, survivable, and lethal aircraft but the pilots flying the aircraft must do so with Viet Nam era equipment. Aircraft designers normally place life support at the end of their list. Even if the aircraft can exceed the limits in altitude or G of conventional aircraft, considerations must be taken to accommodate the pilot.

Pilots flying above 50,000 ft for extended periods of time will be introduced to the physiological problems of decompression sickness (DCS). The longer the pilot remains at the higher altitudes, the chance of DCS increases. DCS is caused following exposure to decreased pressure at high cockpit altitudes. The potential of incapacitation and even death make DCS operationally significant. Sixty minutes at altitude can result in a 10% chance of DCS. The questions currently being asked by the life support experts are: how long will a pilot remain at altitude; will the pilot prebreathe 100% oxygen before operations above 50,000 ft occur; will the flight include repeated transitions to and from the high-altitude arena; and what will the pilot do in a cockpit depressurization situation?

Duration at altitude will depend upon fuel available to the aircraft prior to climbing. Refueling will obviously extend the aircraft's ability to sustain operations above 50,000 ft. Aircraft will climb to the higher altitudes, cruise, descend to the lower altitudes to engage other threats using high-G maneuvers, then climb back up to the higher altitudes to egress home. The transition to and from the high-altitude arena is dynamic and unpredictable due to the nature of the tactical environment.

Prebreathing 100% oxygen denitrogenates the body and can prevent DCS, but it is time-consuming and is, therefore, difficult to employ in fighter aircraft since it would deplete the aircraft's limited oxygen supply. In the case of loss of cockpit pressurization, the pilot will immediately descend to the lower altitudes on all training sorties. No peacetime situation would require the pilot to stay at the higher altitudes. However, in combat the pilot may elect to remain at the higher altitudes for a few more minutes allowing a separation from the threat below before making the descent to a lower altitude. The risks of DCS and hypoxia at the higher altitudes with a cockpit depressurization increases rapidly with time and exposure to the decreased pressure. However, these risks can be offset by prebreathing 100% oxygen, increasing the PPB to higher levels, raising the cabin pressure from 5 PSI to 7 PSI, or wearing pressure suits.

It has already been stated that pressure suits would be impractical in fighter aircraft. However, increasing the coverage of current G suits would increase the protection for the pilot. Increasing PPB levels would also be impractical. Currently, the F-22 uses 70 mmHg to protect the pilot in cases of cockpit depressurization. Breathing 70 mmHg takes practice and is not something one becomes accustomed to after one attempt. PPB is a difficult task and an increase in pressure above 70 mmHg would be extremely uncomfortable and difficult for any pilot. It is therefore not a practical avenue to pursue.

In order to provide protection for the pilot and offset the effects of DCS in high-altitude flight profiles, as well as in cockpit depressurization situations, future aircraft and life-support designs should look to increase aircraft cabin pressure, increase G-suit coverage, and increase oxygen supply to make it practical to prebreathe.

Summary

The F-22 will fly and fight in the advanced radar network and dense surface-to-air missile environments of combat throughout the world. It will use a first-look, first-shot, first-kill capability to detect and destroy enemy fighters today and tomorrow. The F-22 is being developed to counter the increasing sophistication and threat of hostile air superiority forces around the world. Its predecessor, the F-15, entered the Air Force inventory in 1975. Threats that the F-15 can no longer counter will be defeated by the lethal and survivable F-22, with its balance of increased speed and range, enhanced offensive and defensive avionics, and reduced observability. Emphasis on reliability, maintainability and other effectiveness factors will keep the fighter flying in the harshest combat conditions with quick combat servicing.

Aircraft will use high altitudes, high speeds, and stealth to transition through SAM defenses, or climbing to increase the WEZ for their A/A missiles. As engine performance continues to advance, propelling aircraft higher in altitude, there will be a continued drive to exploit the upper atmosphere to take advantage of the high ground. The advances in technology have made quantum leaps in increasing the survivability and lethality of aircraft through advances in stealth and integrated avionics. However, these advances in aircraft design have placed demands on current life support equipment that cannot keep up. Life-support experts have been working hard to provide protection for the pilot since WW I. Future aircraft will continue to challenge them to stay ahead of the physiological problems encountered with high-altitude operations. As aircraft continue to push the extremes of the envelope, aircraft designers and life-support experts must work together to allow the pilot to survive and operate in the hostile environment of high G, upper atmosphere, as well the surface and air threats looking to shoot the aircraft down.

Discussion

MAJ NEUBECK: In summary, a combination of speed, stealth, and high-altitude operations eliminates many threats of interest and reduces the engagement envelope of many others, allowing an aircraft such as the F-22 to transit heavily defended areas without being targeted. The high ground also allows an aircraft to achieve better missile performance than an adversary stuck in lower altitude regimes. Aircraft with high-altitude performance engines will have the advantage of sanctuary above the contrails.

Propulsion technology is advancing to support hypersonic flight regimes above Mach 5 and beyond for aircraft that will transit to and from the battlefield using the upper atmosphere. Current weapons have a hard time maneuvering in the thinner air environment, but with the advancement of thrust vectoring, propulsion and guidance systems, the capability to launch these weapons from very high altitudes to intercept even higher altitude targets will be a reality by the turn of the century.

COL. SHERMAN: Are we going to be reviewing the physiological consequences of exposure to 60,000 or 65,000 feet or higher? There's quite a difference between 60,000 and 70,000 and 80,000 feet.

DR. PILMANIS: We selected 60,000 feet as a starting point and plan on discussing incrementally higher altitudes.

LT. COL. DEMITRY: The users are looking for the cost-to-benefit relationship for every 2,000 or 3,000 feet. I think all would agree that every little change in altitude above 60,000 feet will make a big difference. At these altitudes, it's actually more important to the physiologist than it is to the engine manufacturer who can push the aircraft another 4,000 to 5,000 feet just by decreasing weight or increasing thrust. It's a much more linear relationship for them than it is for a biosystem. One of the modeling tools in the strategic planning process is: how much more is it going to cost me in terms of protection with every 5,000 feet? I don't think we're going to be able to give you much more of an altitude range, unless you can, Maj. Neubeck.

MAJ. NEUBECK: No, I really can't give you a better number. The aircraft will go in excess of 60,000 feet.

LT. COL. DEMITRY: The information can be used in other applications besides the F-22.

COL. STORK: There tends to be increased emphasis on long-range strike and the employment of standoff weapons. How do these concepts change air superiority requirements and crossing the Forward Edge of the Battle Area (FEBA)? Are we going to have to cross the FEBA and be concerned about the SAMs as much in the future as we have in the past?

MAJ. NEUBECK: I think we'll always be required to have the capability of long-range strike. The job of the B-2 and the F-117 is to go beyond the FEBA. We're going to need to take out the SAMs for conventional aircraft to go beyond that area. As far as air superiority having to go that far, wherever the Army goes, that's where the air/ground battle will be. I think we're always going to need the capability to go to deep interdiction based on whatever threat we're trying to put down. Based on stealth alone, the F-22 can get through a lot of SAMs. If you add high speed and high altitude, more of the SAMs will be eliminated and the capabilities of quite a few more will be reduced. So, stealthy aircraft, flying faster at higher altitudes, will allow you to more safely penetrate a greater number of SAM belts.

COL. STORK: Then, should most of our emphasis be placed on the air superiority role, since most other aircraft are not going to have the need to be above 60,000 feet?

MAJ. NEUBECK: There are few threats that can get to that altitude, and they are limited in what they can carry. Most can't get there with their full complement of air-to-air ordnance. The threat that we see right now at 60,000 feet is not an air-to-air threat, but taking out an enemy reconnaissance aircraft. Except for the MIG-25 and -31, there is no air-to-air threat at high altitude.

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DR. ACKLES: Is this a combination of high G and high altitude or just high altitude?

MAJ. NEUBECK: You really can't pull very many Gs at high altitude.

DR. ACKLES: I know, but what is the aircraft capability at low altitude?

MAJ. NEUBECK: It's a 9G aircraft. You have an aircraft that can transition to high altitude in a tactical situation. We may need to go high and fast over a SAM belt to engage the air-to-air threat. So you've got an aircraft that has the engine performance to go high and fast and you can turn at 9 Gs at low altitudes. It's got the benefits of both.

DR. STOLP: How long, would you say, would you be at altitudes above 60,000 feet? Can you give us a rough idea?

MAJ. NEUBECK: You can get above 50,000 feet with supercruise and military power, but to remain there you've got to select afterburner and that's going to cost you fuel. However, you're obviously not burning as much fuel at the higher altitudes as you are the lower altitudes. Its difficult to give you a timeframe, because you can carry additional fuel and sustain operations for quite a long time above 50,000 feet. If we are just considering getting through a SAM belt, employing weapons above the contrails or descending through the contrails to engage other aircraft, you would probably not be at those altitudes very long. The initial high-altitude run that provides a tactical surprise against the air-to-air threat is what we really need to look at.

COL. SHERMAN: It is not how long you're going to be above a certain flight altitude that is important, but whether the cabin pressure is maintained. What we're concerned about is loss of pressure at these higher altitudes and what actions we need to take after the decompression. Do we tactically need to remain at the higher altitudes after loss of pressure or can we roll out and let our partners continue the fight? In the high-altitude reconnaissance business, the aircrew are required to remain at altitude. So I think the key is that we're not concerned how long the pilot stays at what altitude, as long as the cabin pressure is maintained. What we're concerned with is loss of pressure, which hopefully is rare, what we need to do at that point and how long are we going to have to stay there. In my opinion, that should be the driving force for this gathering.

LT. COL. DEMITRY: The engine-specific fuel consumption will go up exponentially if we go into afterburner, so that would decrease time on station for the aircraft. As far as loiter capability, can the aircraft sustain extended periods of operation at high altitude?

MAJ. NEUBECK: That would be difficult to do because you're now talking about a lot of turns in a specific location to stay in a geographic area.

LT. COL. DEMITRY: Then you don't have the turn capability.

MAJ. NEUBECK: You're talking about a fairly large turn which would cut down your time on station.

LT. COL. DEMITRY: Okay, so you really don't envision going high to reduce fuel consumption in order to stay for an extended period, even though you're not in a supercruise regime?

MAJ. NEUBECK: No, you've got to go to burner to remain higher. It's going to cost you.

DR. ACKLES: I'm assuming that the F-22 has a 5 PSI cockpit differential?

DR. SEARS: Yes.

DR. MOON: As a pilot, could you comment on use of or acceptability of a fully pressurized suit?

MAJ. NEUBECK: I would be interested in looking at a pressure suit if it didn't impair my visibility, provided me with protection to 9 Gs, reduced heat stress, is comfortable and functions with all the newer life-support and display systems. We're currently dealing with night-vision goggles and helmet-mounted display systems that all

add weight to the head. These systems must now be integrated into a fully enclosed helmet. It would be a challenge to design such a system. We're just now fielding COMBAT EDGE equipment and ATAGS will be fielded in the next few years. We've got a long way to go, just for the basic stuff we have right now.

DR. BOMAR: You've indicated that we have several aircraft in the inventory that are capable of going above 50,000 feet. We've basically got a 47,000 foot oxygen system in those aircraft. From your F-15 experience, what do you think the current risk is in terms of pilots now flying at or above 50,000 feet?

MAJ. NEUBECK: The threat right now, going to altitudes of 50,000 feet and above? I would expect that if an engine or an ECS fails, the guy is probably going to become hypoxic.

DR. BOMAR: Is 50,000 feet a practical altitude for maneuvering now in an F-15?

MAJ. NEUBECK: You bet. You have that in either the F-16 or F-15; anyone flying a fighter that can get high will do so. You have a pilot that knows the operational limit is 50,000 feet, but if he believes that the guy on the other side won't detect them at a little higher altitude, he'll go higher.

DR. BOMAR: That is very important. The operational limit for the oxygen system in current aircraft is 47,000 feet; the emergency limit is 50,000 feet. The oxygen system that we have in current aircraft provides very poor protection at 50,000 feet. The ECS systems are not as robust in these aircraft as you're planning for the F-22. If people are now flying above the operational ceiling in current aircraft, and we design a system for 60,000 feet, can we expect that they will routinely fly above 60,000 feet in future operations? If so, we are leaving them exposed to a hazardous condition by a lack of knowledge regarding future operational scenarios.

MAJ. NEUBECK: You have guys flying at 50,000 feet every day, so they're there now.

DR. MACMILLAN: It's the cabin altitude that is important following the decompression, i.e., aerodynamic suction will likely cause the cabin altitude to be higher than the flight altitude after loss of pressure. That should be considered in any decision establishing protective requirements.

DR. ACKLES: As a research community, we frequently have had difficulty getting involved in aircraft development programs in a timely manner. The F-22 has been one of the best programs to be involved in, from the standpoint of crew protection and integration, but we need to be prepared to train the aircrews for the risk(s) that they may be exposed to at these higher altitudes. Colonel Sherman directs physiological training for the USAF and before we have an initial operating capability for this aircraft, we need to be able to train the aircrews like the UK has done for years. So we need to be involved in the training plan for the weapon system as early-on as possible.

PROF. ERNSTING: I'd just like to go back to the question that was asked earlier, which I think is of great concern to us all, what do you propose to do in this airplane if you do lose pressure at high altitude? If you actually lose cabin pressure at high altitude, do you propose to come down below 40,000 feet, or 30,000 feet in the operational scenario?

MAJ. NEUBECK: I believe you're going to descend to that level necessary to maintain consciousness, even if the lower altitude will expose you to an enemy threat.

PROF. ERNSTING: Yes, so you're letting the aeromedical considerations drive the maximum altitude to which you descend on loss of cabin pressure?

MAJ. NEUBECK: That's really a driving factor for all of our planes.

COLONEL MARLOWE: What kinds of loiter times are you expecting at 60,000 feet?

MAJ. NEUBECK: You're probably not going to loiter at 60,000 feet. You're probably going to transition over a high-threat environment at 60,000 feet, so your time at that altitude will not usually be very long. I really don't

have any numbers for you, but whatever that transition time is for maybe 300-400 miles. When someone brings up the word "loiter", that means I'm in a fixed geographic location directed by AWACS and I have to maintain a station. In that case, I'm probably going to be around 30,000 feet where I can conserve my gas and not hamper aircraft performance.

COL. WORKMAN: I have brought with me the next generation full-pressure suit, the S1034, that we are bringing into the inventory for the U-2 community. I think it will give the group an opportunity to frame some thoughts relative to Maj. Neubeck's comments on visibility, weight, and maneuverability, and just see what the current state of pressure suit technology is today.

USAF Life Support Development Efforts--1995

Martin J. Clement, Lt Col, USAF

Introduction

This paper highlights the activities in the USAF Life Support Systems Division of the Human Systems Center at Brooks AFB, Texas. Our primary mission is to develop and field the next generation of aircrew life-support equipment. Each of the funded programs under development, as well as new efforts awaiting user direction and funding, will be detailed. The focus, however, is on the physiological aspects of several of our key programs. In the Life Support Systems Division, we have four integrated product teams, each working user-supported and funded development initiatives. These programs are: COMBined Advanced Technology Enhanced Design G-Ensemble (COMBAT EDGE), Universal Water Activated Release System (UWARS), Night Vision Systems, and Active Noise Reduction. Each of these programs is in a different stage of the development process. Following the information on the four major programs is a brief outline of projects in our advanced projects and planning area. A discussion of our role as prime USAF integrator of life support equipment, to develop and field equipment that meets multiple needs simultaneously, wraps up this brief expose.

Part 1: Programs in Engineering and Manufacturing Development (EMD)

Our highest visibility program, COMBined Advanced Technology Enhanced Design G-Ensemble (COMBAT EDGE), is being fielded in USAF tactical fighters (F-15s and F-16s) to reduce the occurrence of G-induced Loss of Consciousness (GLOC). COMBAT EDGE has its roots in the Tactical Life Support System (TLSS) demonstration/validation program in the mid-eighties. In 1988, following numerous F-16 GLOCs, the Tactical Air Command (now Air Combat Command) commander ordered expedited fielding of certain components of the TLSS program. This assemblage of equipment acquired the moniker of COMBAT EDGE. Deployment to all USAF F-16s is complete, with the modification and outfitting of F-15s to start in September of this year. COMBAT EDGE provides positive pressure breathing that, in lay terms, primarily makes it easier to perform an anti-G straining maneuver. This effectively reduces fatigue and allows the pilot to concentrate on fighting. Components of the system include both aircraft and manside hardware. The aircraft installation includes a new oxygen regulator connected to the high-flow G-valve with a sense line. This controls the pressure ratio between the lower G-garment and the oxygen flow pressure to the mask and the counterpressure vest. The manside hardware includes a new high-pressure mask, counterpressure vest, an integrated terminal block for the hose connections, and occipital helmet bladder to tension the mask to the face under positive pressure breathing. Based on operational test and evaluation comments, the mask is undergoing design changes to improve field of view and valsalva. Currently, COMBAT EDGE is not hardened to operate in a chemical warfare environment. One of our new project efforts with the Armstrong Lab is to demonstrate the concept of chem-hardened PBG. More on that later in the new projects area. The US Navy is in the final stages of its evaluation to outfit F/A-18s with Navy COMBAT EDGE. In the early stages of their assessment we were working with them to outfit their test aircraft. The Navy COMBAT EDGE is essentially the USAF system with the addition of a chest-mounted regulator and the new Navy version of an increased coverage lower G-garment. The counter pressure vest will be part of the F-22 ensemble.

Another of our small programs, but for us a major effort, is the Universal Water Activated Release System. This is a water-activated parachute riser release device for the incapacitated or unconscious aircrew. The term universal applies to the type of parachute fittings in the USAF inventory. UWARS replaces a larger device that is not compatible with all USAF fittings, does not have a built in test capability, and is nearing the end of its useful service life. During the development of this state-of-the-art pyrotechnic device, there were numerous

engineering challenges to overcome: battery power requirements and characterizations to meet the environmental requirements, and "leading edge technical" circuit problems. These were resolved by aggressive management and contractor oversight. On both of these issues we led the contractor to the design fix and helped engineer the solution. The system is in the final stages of operational testing and, following successful completion, we expect to start producing them with deliveries in 1996.

Our flagship development program is the Night Vision Systems effort. The purpose of this program is to engineer and field the first US ejection compatible image intensifying device for fast moving aircraft. Most USAF MAJCOMs are involved, as well as the US Navy and Marines. The equipment will also be targeted for use in helicopters and transports for long-duration missions. Our strategy called for evaluating two contractors with different approaches to translating the intensified image to the eyes. The Type I (direct view) and Type II (combiner lens arrangement) systems both went through the design phase and subsequent downselect evaluation. The direct view system was selected and is continuing in the next phase of our program, which consists of development test and evaluation. Benefits from this new device will be: lighter weight than currently fielded, complete logistics support system, and included ejection compatibility. This last phrase is our most difficult concept to address--ejection compatibility. Trying to define the parameters of this interface is difficult at best. Considerations include safe head center-of-gravity positioning, neck strength of men and women, timing for release of the device prior to ejection, and subsequent travel of the device when the windblast effect occurs. We are in the process of establishing a safety advisory group of recognized experts in the appropriate disciplines to help us get our arms around this issue. Initial operating capability is slated for 1998.

The last of our programs formally in the engineering and manufacturing development category is the Active Noise Reduction (ANR) system. Originally a Strategic Air Command requirement for bomber and tanker aircraft in the late 1980s, but following the subsequent consolidation of the MAJCOMs, it was dropped by Air Combat Command. In 1991 our special operations command, AFSOC, re-wrote the requirements documents for their helicopters and transport aircraft. ANR program goals are to improve communications through better speech intelligibility, minimize fatigue from noise exposure, and reduce temporary and permanent hearing loss. Technically, the device senses the noise pressures in the earcup and generates an out-of-phase shifted signal to reduce the noise levels to the ear. It provides attenuation in the lower frequency ranges (below 1000 hertz). We are anticipating widespread user interest following initial fielding, when the intangible benefits will be recognized. The F-22 helmet incorporates the use of this device and other services are looking at this and other noise reducing systems. Initial capability for the system will be in 1996. A lightweight helmet is a necessary starting point for ANR use in fighters, as this system, in its current configuration, adds a half of a pound to the helmet weight.

Part 2: Projects in Early Stages of Development

Part of our organization works with the labs and users on promising technologies to address anticipated user needs or to complete the development of new capabilities for increased combat effectiveness. These activities are more properly addressed as "projects" as we await requirements documents, funding, and direction to formally proceed as a program. This list of projects includes: the Advanced Technology Anti-G Suit (ATAGS), COMBined Advanced Technology Acceleration/ Chemical Ensemble (COMBAT ACE), laser eye protection (LEP), and the ACES II ejection seat continuous improvement program.

Another item with family lineage from the Tactical Life Support System (TLSS), which holds significant promise, is the full-coverage lower anti-G suit, ATAGS. At the time COMBAT EDGE was pulled out of TLSS, the anti-G suit was still in need of refining. ATAGS was originally conceived to work in concert with positive pressure breathing. It complements COMBAT EDGE extremely well, providing better return of blood from the lower extremities. This enhances positive pressure breathing capability to keep the oxygenated blood at brain/eye level. Over the last several years it has gone through engineering updates, an early operational assessment in F-15s and F-16s, and hundreds of hours of centrifuge tests. The project officially transitioned from the lab to our program office in early 1993 awaiting user requirement documentation and funding. It is projected to replace the current anti-G suit, which has been in the inventory since the end of World War II. The current plan is to finish the

engineering development "in house," then go to industry competitively to produce anti-G suits for operational test, followed by production. We are expecting to kick-off the program, with a user requirement, funding, and direction, sometime in 1996. ATAGS is the lower body anti-G suit for the F-22.

COMBAT ACE, yet another concept from the TLSS early development program, is to provide chem-defense capability for positive-pressure-breathing-equipped aircraft. Now that COMBAT EDGE has been fielded to address G-Induced Loss of Consciousness, the next logical step is to harden it for chemical warfare. The chest counter-pressure garment is being modified for chemical warfare by the F-22 program office. We are working through the lab on a task order contract with Boeing to resolve the deficiencies of the TLSS chemical-defense ensemble. This potential system may have future application in the F-22 as well as the F-15s and F-16s. There is no user direction, requirement, or funding for this project, but there is significant interest, as combat-ready units have recognized the need for PBG chemical-defense protection.

The laser eye protection issue has been around for the USAF since the late 1980s. Early hardware, fielded under urgent requirements in 1987, does not integrate well with the glass cockpit displays we have today. It is also out of production, and does not protect against all projected threats. We have been working closely with the Armstrong Lab and the operators who are interested in a quick fix to the instant "buddy lasing" problem. The technology most readily available is the absorptive dye-based technology. Should an urgent need be established by the users, we would be initiating a quick turn program to meet their needs. In the long term there are other technologies being tracked, holographic and agile protection. Laser eye protection is similar to chemical defense: as soon as the user validates the threat and makes this a requirement, and even more importantly provides the funding, we are ready to do the programmatics to field the systems.

The last area in which we are involved is improving the ACES II ejection seat. There are many safety features that could be explored for incorporation into the seat through retrofit. However, these upgrades must be thoroughly designed and tested. Recent mishaps have highlighted the need to address this critical life-saving system with the latest in technology to improve its capability. This seat is going to be in the inventory for a long time and needs upgrades to provide adequate safety margin for our high-performance aircraft. The ACES II is an outstanding seat with a high success rate, but there are still technologies that could be applied to make it a better egress system. The fourth generation work being done in the lab at Wright-Patterson Air Force Base is demonstrating some of these technologies that should be assessed for ACES II retrofit to enlarge the envelope of this seat to more closely match the performance envelope of high-performance aircraft.

Summary

As the lead agency for development of life-support systems equipment for the USAF, we are charged with integrating individual pieces of equipment to meet multiple requirements. Historically, our approach has been to design and develop each piece of equipment as part of an integrated whole, but in a modular fashion--wearing only those items required to accomplish the mission. This is a challenging task given the vagaries of human anthropometry and equipment fitting, lack of acceptance by the users of certain threats, lack of sufficient development and production funding, minimal user interest in something that is not glamorous like a new airplane, and having separate contracts to manage each system. This does not lend itself to ideal integration of hardware, but under the circumstances with minimal interest and funding, there is no other alternative. We are developing a concept for the future that again finds its family roots in TLSS. This composite suite of hardware will meet the needs of the tactical fighter community of the present and future.

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Discussion

CAPT. SCOGGINS: Do you make a distinction between programs versus projects? Are there significant differences?

LT. COL. CLEMENT: Current programs are those efforts where we have established integrated program teams based on user requirements and funding to develop a specific piece of equipment. The projects are not currently user funded and the requirements documents are either in work or being developed.

DR. PILMANIS: For the record, why do you not have a high-altitude protection program?

LT. COL. CLEMENT: Right now the F-22 is the only program I know of, other than the U-2, that has a firm high-altitude requirement. We are working with the F-22 program office who does have an ongoing program for protection to 60,000 feet, but we don't have a requirement for high-altitude protection at this time. They're trying to provide protection for high altitude, high G, thermal and chemical/biological agents. We aren't doing that right now. As regards pressurized systems, we only have requirements for the Combined Advanced Technology Enhanced Design G Ensemble (COMBAT EDGE), Advanced Technology Anti-G Suit (ATAGS) and Combined Advanced Technology Acceleration/Chemical Ensemble (COMBAT ACE) efforts, and mostly for retrofit into F-15 and F-16. We're also working in conjunction with the F-22 program office to supply them equipment for lower altitudes.

Hypoxia Prevention -- Review of Acceptable Compromises Prior to Decompression

John Ernsting, Prof., Air Vice Marshall RAF (Rtd),
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Introduction

The cabins of high-performance agile aircraft are pressurised during flight in order to provide an acceptable pressure and thermal environment for the crew. In these aircraft, hypoxia during flight is prevented by delivering to the respiratory tract gas containing the appropriate concentration of oxygen in relation to cabin altitude. Whilst the use of 100% oxygen during routine flight has certain advantages, it also has disadvantages that have led many air forces to employ breathing systems that provide a mixture of oxygen and air. Furthermore, the recently introduced molecular sieve oxygen concentrator systems generally provide breathing gas containing nitrogen and argon as well as oxygen. Hypoxia is the most immediate and serious hazard that follows failure of the pressure cabin of such an aircraft flying at high altitude. Prevention of hypoxia on exposure to pressure-altitudes above 40,00-43,000 feet requires the immediate provision of breathing gas containing 94%-100% oxygen at an appropriate absolute pressure (7).

Amongst others, the author has reviewed the factors that influence the relationship between the concentration of oxygen in the inspired gas and cabin altitude on several occasions (5, 9, 10, 11). The compromises between the degree of hypoxia and level of pressure breathing when using partial-pressure suits that employ oronasal masks have also been studied extensively (2, 7, 21). Some of the results of these studies have been embodied in standards published by the Air Standardisation Coordination Committee (Air Standards 61/101/6A and 61/101/1C)(1) and by the NATO Military Agency for Standardisation (STANAG 3865) (23). This paper reviews the physiological and operational factors that determine the relationship between the concentration of oxygen in the inspired gas and cabin altitude in agile combat aircraft when the cabin is pressurised.

Most present agile aircraft, and those that are to be introduced into operational service in the next few years, employ a cabin pressurisation schedule in which the maximum differential pressure of 5.0 Lb in^{-2} is operative at aircraft altitudes above 23,000 feet. Several physiological considerations influence the requirement for the composition of the gas to be delivered to the respiratory tract during pressurised flight. In conventional aircraft oxygen systems, the diluent gas is virtually entirely nitrogen since the 100% oxygen from the aircraft store of gaseous or liquid oxygen is diluted with cabin air before delivery to the mask. The performance of presently available molecular sieve oxygen concentrators is such that the product gas contains argon as well as oxygen and nitrogen. The maximum concentration of argon in the product gas does not exceed 5-6%. Laboratory studies (3) have shown that in these low concentrations, argon has no specific physiological effect and can be regarded in this context solely as a diluent gas.

Minimum Concentration of Oxygen to Prevent Hypoxia in the Pressurised Cabin

The concentration of oxygen in the inspired gas must be adequate to prevent significant hypoxia. Ideally, the concentration of oxygen should always be such that the partial pressure of oxygen (PO_2) in the alveolar gas is maintained at or above the normal value associated with breathing air at ground level, i.e., 103 mm Hg (Figure 1). This minimum standard should always be employed where the quantity of available oxygen allows it. Greater economy in the use of oxygen can be achieved by providing a lower concentration of oxygen than that required to maintain an alveolar PO_2 of 103 mm Hg. The review performed in 1978 (9) and subsequent studies (Farmer, personal communication) have supported the conclusion that the minimum acceptable alveolar PO_2 for the crew of
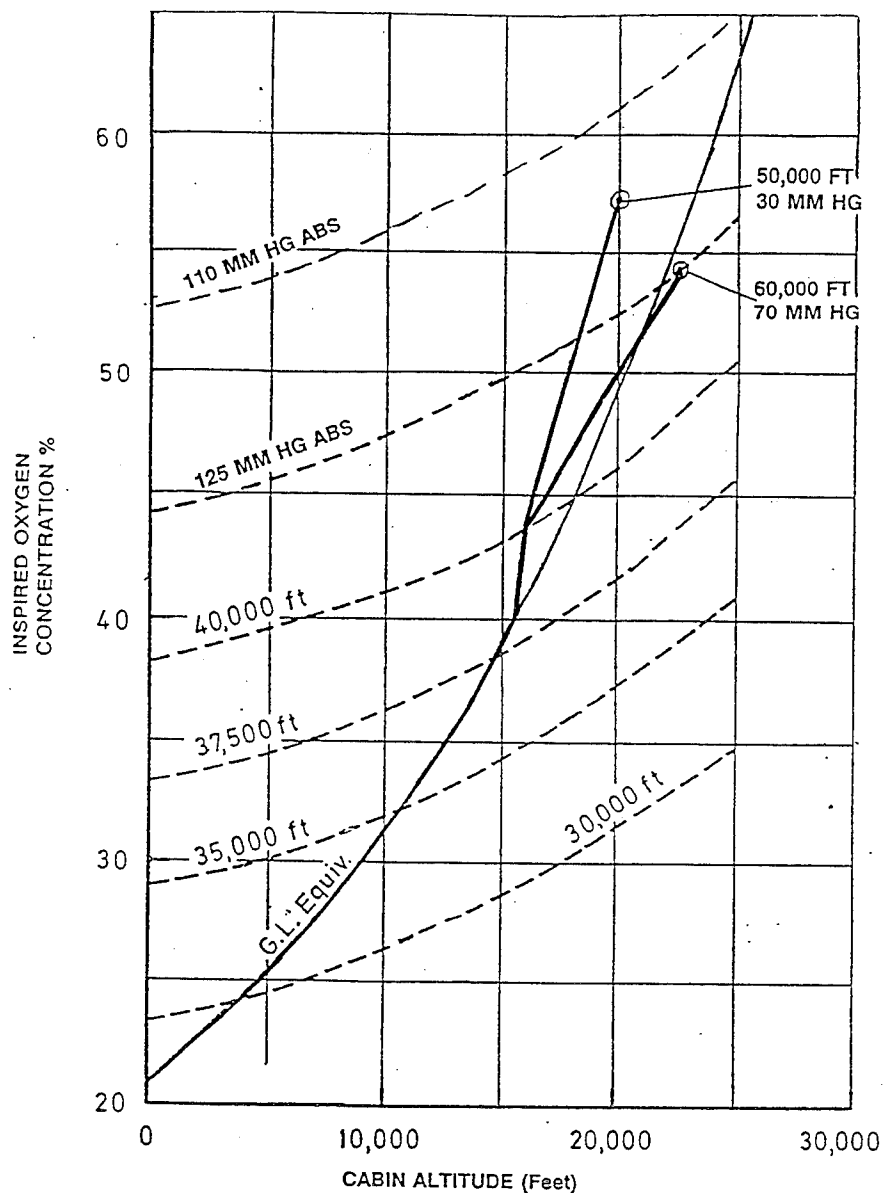
Figure 1. The relationships between the concentration of oxygen in the inspired gas and the pressure-altitude within the pressurised cabin [cabin altitude] required

i.   to maintain an alveolar PO_2 of 103 mm Hg i.e., equivalent to breathing air at ground level [GL Equiv]

ii.   to produce an alveolar PO_2 of 30 mm Hg on an instantaneous decompression from the cabin altitude indicated on the X axis of the figure to the cabin altitude/absolute intrapulmonary pressure indicated by the broken horizontal curves [final cabin altitudes of 30,000, 35,000, 37,500 and 40,000 feet and final intrapulmonary absolute pressures of 141 mm Hg (40,000 feet curve) 125 and 110 mm Hg].

iii. to ensure, with a cabin pressure differential of 5 Lb in^{-2}, that an instantaneous decompression from the cabin altitude indicated on the X axis, the alveolar PO_2 immediately after the decompression will be 30 mm Hg when

    a.   using a pressure breathing system which provides a breathing pressure of 30 mm Hg gauge at 50,000 feet [50,000 FT; 30 mm Hg]

    and

    b.   using a pressure breathing system which provides a breathing pressure of 70 mm Hg gauge at 60,000 feet [60,000 FT; 70 mm Hg]

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an agile combat aircraft is 75 mm Hg, which is the alveolar PO_2 associated with breathing air at an altitude of 5,000 feet. It is now accepted practice (Air Std 61/101/6A (1) and STANAG 3685 (23)) to provide an oxygen concentration that will maintain alveolar PO_2 of at least 100-103 mm Hg at all cabin altitudes when the cabin is pressurised.

Many aircraft molecular sieve oxygen concentrator systems include one or more oxygen sensors that monitor the concentration or partial pressure of oxygen and provide a signal in the event that the oxygen concentration of the product gas falls below an acceptable level. In order to avoid spurious warning of an inadequate oxygen supply and yet ensure an early warning of possible impending hypoxia, it has become accepted practice to set the oxygen sensors to maintain the alveolar PO_2 between 75 and 103 mm Hg (i.e., equivalent to breathing air at an altitude between ground level and 5,000 feet) when the concentration of oxygen in the product gas is between the limits required.

Minimum Concentration of Oxygen Required
to Prevent Hypoxia on a Subsequent Rapid Decompression

A further important factor that influences the relationship between the minimum concentration of oxygen in the inspired gas and cabin altitude when the cabin is pressurised is the requirement to prevent impairment of performance due to hypoxia following a decompression of the pressure cabin at high altitude (6, 9, 13). If the inspired gas breathed with the cabin pressurised contains a significant concentration of nitrogen, then hypoxia may well follow a rapid decompression to altitudes above 30,000 feet, even if 100% oxygen is breathed from the instant at which the decompression occurs, and the intrapulmonary pressure does not fall below 125 mm Hg absolute. The fall of the total pressure of the alveolar gas produced by the rapid decompression will produce a concomitant reduction of the alveolar PO_2 that will increase after the decompression as the 100% oxygen in the inspired gas progressively reduces the concentration of nitrogen in the alveolar gas. Extensive experimental studies (9, 13) have demonstrated that if the alveolar PO_2 is reduced to below 30 mm Hg in these circumstances even for only a few seconds, then the consequent transient hypoxia will produce a significant impairment of mental and psychomotor performance. If the magnitude of the area enclosed between an alveolar PO_2 of 30 mm Hg above and the time course of the alveolar PO_2 below exceeds 140 mm Hg sec, then the individual will almost certainly (95% chance) become unconscious (8). Between the limits of 0 to 140 mm Hg sec, the decrement of performance at a choice reaction task was found to be proportional to the magnitude of the area bordered by PO_2 of 30 mm Hg above and the time course of the alveolar PO_2 below (8). The breathing gas delivery system should therefore prevent the alveolar PO_2 falling below 30 mm Hg during and subsequent to a rapid decompression.

The major factors determining the minimum value of the alveolar PO_2 immediately after a rapid decompression are the initial and final absolute pressures of the alveolar gas and the composition of the gas breathed before and after the decompression. Assuming that 100% oxygen is delivered to the respiratory tract immediately after the decompression occurs, the alveolar PO_2 can be prevented from falling below 30 mm Hg by ensuring that the gas breathed before the decompression contains an adequate concentration of oxygen. Assuming that no gas is inspired during a rapid decompression and that the magnitude of any exchange of oxygen between the alveolar gas and the blood flowing through the lungs will be insignificant, then the alveolar PO_2 at the end of a rapid decompression is given by equation 1.

$$\text{Final PAO}_2 \ = \ \text{Initial PAO}_2 \ x \ \frac{(PF - 47)}{(PI - 47)} \qquad \qquad \text{Equation 1}$$

where:

Initial PAO$_2$	=	Alveolar PO_2 before the rapid decompression
Final PAO$_2$	=	Alveolar PO_2 immediately after the rapid decompression
PI	=	Total alveolar gas absolute pressure before the rapid decompression in mm Hg
PF	=	Total alveolar gas absolute pressure immediately after the rapid decompression in mm Hg

Equation 1 may be used to calculate the value of the alveolar PO_2 required before the rapid decompression, for given values of the initial and final pressures of the alveolar gas, to produce an alveolar PO_2 of 30 mm Hg immediately after the rapid decompression (10). It is then possible to calculate, using the Alveolar Gas Equation, the concentration of oxygen in the inspired gas required to produce the calculated alveolar PO_2 before the rapid decompression, having made assumptions as to the values of the alveolar PCO_2 and the respiratory exchange ratio. The concentrations of oxygen required in the inspired gas to produce an alveolar PO_2 of 30 mm Hg immediately after a rapid decompression from a given initial cabin altitude to a given final cabin altitude [total absolute alveolar gas pressure at final cabin altitudes above 40,000 feet] are indicated by the interrupted curves of Figure 1. The relationship between initial cabin altitude and the final cabin altitude is determined by the pressurisation schedule of the cabin of the aircraft. The final alveolar gas pressure (PF) is determined by the safety pressure/pressure breathing characteristics of the breathing gas delivery systems. Thus the curve relating the minimum concentration of oxygen in the inspired gas to cabin altitude before a decompression required to prevent the alveolar PO_2 falling below 30 mm Hg immediately after the decompression will depend upon the cabin pressurisation schedule of the aircraft and the safety pressure/pressure breathing characteristics of the breathing gas delivery system.

The minimum inspired oxygen concentration-cabin altitude curves for two commonly used pressure-breathing systems employed in an aircraft with a cabin pressure differential of 5 Lb in^{-2} at aircraft altitudes above 23,000 feet are presented in Figure 1. Both of these pressure-breathing systems commence pressure breathing at a cabin altitude of 40,000 feet and deliver oxygen at an absolute pressure that falls linearly with the reduction of environmental pressure at altitudes above 40,000 feet. One system, comprised of a pressure-breathing mask without counterpressure to the body, employs a breathing pressure of 30 mm Hg at 50,000 feet, which provides an intrapulmonary pressure of 117.5 mm Hg absolute at 50,000 feet (7). The other system, comprised of a pressure breathing mask with counterpressure to the trunk and lower limbs, employs a breathing pressure of 70 mm Hg at 60,000 feet, which provides an intrapulmonary pressure of 124 mm Hg absolute at 60,000 feet (7). It may be seen from Figure 1 that the minimum concentration of oxygen required in the inspired gas to prevent significant hypoxia being induced by the rapid decompression is greater than that required to maintain an alveolar PO_2 of 103 mm Hg in the steady state at cabin altitudes above 16,000 feet. The concentration of oxygen required in the inspired gas at cabin altitudes above 16,000 feet is greater with the pressure breathing system that employs a breathing pressure of 30 mm Hg at 50,000 feet than the system that employs a breathing pressure of 70 mm Hg at 60,000 feet. Increasing the differential pressure of the cabin above 5 Lb in^{-2} will shift both these curves in Figure 1 to the left (12).

Recent studies have confirmed the magnitude of the transient hypoxia associated with the alveolar PO_2 falling below 30 mm Hg on rapid decompression and the value of increasing the concentration of oxygen in the inspired gas breathed before a rapid decompression (16). It is now accepted practice (Air Std 61/101/6A(1) and STANAG 3865(23)) to require that the minimum concentration of oxygen in the inspired gas shall be such as to prevent the alveolar PO_2 falling below 30 mm Hg on rapid decompression of the cabin when 94-100% oxygen is delivered immediately after the decompression.

The importance of the immediate delivery of 100% oxygen to the respiratory tract on rapid decompression to a final altitude exceeding 30,000 feet is recognised in the current standards (1, 23). These require that the concentration of oxygen in the inspired gas shall rise to at least 94% when no more than 0.6 litre (ATPD) of gas has been inspired following the beginning of the rapid decompression. This requirement is met by conventional pressure demand air dilution regulator systems provided that the volume of the hoses between the outlet of the regulator and the inlet valve of the mask is less than 0.6 litre (a requirement that is easily fulfilled when the pressure demand regulator is mounted either on the ejection seat or the torso of the occupant). Special attention has to be paid to this requirement in molecular sieve oxygen concentrator systems even when a source of 100% oxygen is selected automatically on a rapid decompression as the cabin altitude exceeds 25,000 feet. Significant quantities of nitrogen can be trapped in such systems in the pipework between the Emergency/Back Up Oxygen Supply and the pressure demand regulator. Recent studies have confirmed the requirement to ensure that the oxygen concentration in the inspired gas rises to 100% before 0.6 litre (ATPD) of gas has been inspired during and after a rapid decompression. Failure to deliver 100% oxygen until 1.0 - 1.1 litre (ATPD) of gas had been inspired produced unacceptable transient hypoxia on rapid decompression to an absolute pressure of 122 mm Hg (16).

Breathing 100% oxygen before flight or from the commencement of flight at cabin altitudes up to at least 20,000 feet significantly reduces the incidence of severe venous gas emboli and overt decompression sickness following decompression to a higher altitude (27). Although the symptoms of decompression sickness occur very rarely in current combat aircraft operations, the possibility of extended duration flights at cabin altitudes above 15,000-18,000 feet has led to the suggestion that pilots of combat aircraft should breathe 100% oxygen throughout flight in order to reduce the hazard of serious decompression sickness arising either during high-altitude flight or after decompression of the pressure cabin at high altitude (27). Whilst there is no doubt that breathing 100% oxygen throughout flight would reduce the probability of decompression sickness occurring at high cabin altitudes or following rapid decompression of the cabin at high altitude, breathing 100% oxygen produces lung collapse on exposure to +G_z accelerations and delayed otitic barotrauma. Furthermore, the incidence of significant symptoms of decompression sickness at cabin altitudes up to 20,000 feet in combat aircraft is extremely low. Present evidence also suggests that if immediate descent is undertaken to altitudes below 25,000 feet following decompression of the cabin, serious decompression sickness will occur only rarely, even if the gas breathed prior to the decompression contains 45-50% nitrogen. Indeed, this assumption is one of the bases of many "get-me-down" partial-pressure suit systems (7, 26). The balance between the requirement to avoid the disadvantages of breathing 100% oxygen throughout flight and the possibility of developing significant decompression sickness either with the cabin pressurised or following loss of cabin pressure, varies markedly with the cabin pressurisation schedule of the aircraft and the flight profiles to be employed operationally. Whilst conventional oxygen systems using gaseous or liquid oxygen stores in the aircraft can provide 100% oxygen throughout flight (possibly limiting the duration of a sortie), several of the molecular sieve oxygen concentrator systems now in service or to be fitted to combat aircraft to be introduced into service towards the end of this decade will not provide nitrogen-free breathing gas throughout flight.

Maximum Concentration of Oxygen

Breathing a gas mixture containing a high concentration of oxygen during flight in agile combat aircraft has two important disadvantages. It produces acceleration-induced atelectasis and delayed otitic barotrauma. In 1956, setting the pressure demand regulators in fighter aircraft of the Royal Air Force to deliver 100% oxygen and not oxygen diluted with air gave rise to symptoms during and especially after flights in which the crew had been exposed to +G_z accelerations when using G trousers (4). The symptoms of the condition were attacks of dry coughing accompanied often by a sense of difficulty of breathing, or less frequently by substernal pain and tightness in the chest. The coughing was usually provoked by attempts to take a deep breath either in flight or, more frequently, on standing up in the cockpit after flight. The cough and difficulty in breathing lasted from a minute or so to repeated attacks over a period of 10-30 min. Field studies (4, 19) showed that 80-85% of pilots developed the condition with symptoms during or following flights in which 100% oxygen was breathed and +G_z manoeuvres above 3-4G were performed. Chest radiographs revealed marked collapse of the basal parts of the lungs (19). The lung collapse remained after return to +1G_z until the individual took a deep breath and/or coughed. Although radiological signs of lung collapse occasionally remained beyond 24 hours after the exposure to +G_z acceleration breathing 100% oxygen, the chest X-ray usually returned to a normal appearance within 10-30 minutes after flight. Acceleration atelectasis is associated with a reduction of the Vital Capacity that is recovered by taking deep breaths. Exposure to 4G acceleration for 75 seconds reduced the Vital Capacities of a group of subjects by 40 - 60% (17).

Extensive laboratory studies using man-carrying centrifuges (14, 15, 17, 22) have confirmed that the causative factors of acceleration atelectasis are exposure to +G_z accelerations greater than 3-4G and breathing 100% oxygen, and that the degree of lung collapse and the intensity of the symptoms are greatly increased by inflation of the G trousers. The mechanism is absorption of gas from non-ventilated alveoli in the lower parts of the lungs. The ventilation of these alveoli ceases on exposure to +G_z acceleration as the increased weight of the lung above compresses the lower parts of the lung, closing the small and intermediate sized airways. This process is accentuated by the inflation of the abdominal bladder of the G trousers. When the gas breathed before the exposure to +G_z acceleration is air, the presence of a high concentration of nitrogen in the non-ventilated alveoli

maintains the patentcy of the latter whilst the increased accelerative force is operative and ventilation of the alveoli recommences on return to 1G. If, however, the gas breathed before the exposure to +G_z acceleration is 100% oxygen so that the concentration of nitrogen in the alveoli is very low, the blood flowing through the non-ventilated alveoli rapidly absorbs all the gas trapped in the alveoli and surface forces maintain the alveoli in the collapsed state after the return to 1G until they are reopened by a deep inspiration and coughing. Rahn and Dale (25) demonstrated in animal studies that the rate of absorption of gas from non-ventilated alveoli is increased sixty times when 100% oxygen is breathed instead of air before the cessation of ventilation of the lungs. The presence of a significant concentration of nitrogen that has a much lower solubility in blood than oxygen and carbon dioxide acts as a brake on the absorption of gas from the non-ventilated alveoli. Mixed venous blood continues to flow through the collapsed lungs and thus the condition produces a right-to-left shunt, the magnitude of which varies with the degree of acceleration atelectasis. Thus a moderate exposure to 4G for 75 seconds whilst breathing 100% oxygen induced a right-to-left shunt of 20-25% of the cardiac output in resting subjects (18). Whilst such a shunt may be of little significance with respect to the oxygen content of the arterial blood for as long as 100% oxygen is breathed at low altitude, it would produce a very significant decrease in the arterial oxygen saturation if the alveolar PO_2 was reduced to below 100 mm Hg by a subsequent exposure to high altitude.

Although no long-term deleterious effects have been found in aircrew who have developed acceleration-induced atelectasis repeatedly in flight, many air forces consider that the chest discomfort that it produces and the potential hazard to flight safety of coughing in flight make acceleration atelectasis unacceptable. Extensive flight and laboratory trials conducted by the Royal Air Force in the early 1960s (17, 18) and recently repeated by the United States Air Force (20) demonstrated clearly that acceleration atelectasis does not occur if the concentration of nitrogen in the gas breathed before and during the exposure to the sustained acceleration does not fall below 40%. The study conducted by Haswell et al (20) also demonstrated that argon in the concentration expected in the product gas with oxygen and nitrogen from a molecular sieve oxygen concentrator is as effective as nitrogen in preventing acceleration atelectasis. An animal study of the rate of absorption of gas from non-ventilated alveoli at reduced environmental pressures up to a pressure-altitude of 25,000 feet suggested that the concentration of nitrogen required to prevent acceleration atelectasis at altitude is also about 40% (6). Flight experience at cabin altitudes up to 20,000 feet confirmed this finding. The Royal Air Force has required, therefore, since 1960, that the concentration of oxygen delivered by aircraft oxygen systems when in the air dilution mode does not exceed 60% at cabin altitudes below 20,000 feet. In practice, the need for economy in the use of oxygen in high-performance combat aircraft has led to the use of air dilution demand regulators in most NATO air forces including the Royal Air Force and the United States Air Force. The maximum concentration of oxygen delivered by these regulators in the air dilution mode at cabin altitudes up to 20,000 feet has been less than 60% and acceleration atelectasis has not occurred. The United States Navy has employed 100% oxygen in many of its combat aircraft over this period in order to enhance protection against toxic fumes in the cabin and against drowning on parachute descent into water. US Navy aircrew report the symptoms of acceleration atelectasis in flight, (Baker--personal communication). The Royal Air Force requirement for an adequate concentration of nitrogen in the inspired gas at cabin altitudes up to cabin altitudes of 16,000-20,000 feet was confirmed during the specification of the molecular sieve oxygen concentrator system for the RAF Harrier GR Mk 5 aircraft. Test flights in the development USN AV-8B aircraft in which the molecular sieve oxygen concentrator system delivered 94% oxygen resulted in acceleration atelectasis when moderate levels of +G_z were experienced.

Breathing 100% oxygen, especially if it is associated with even moderate ascent to and descent from altitude, is followed in the vast majority of individuals by the development of delayed otitic barotrauma. On waking from a night's sleep, following flights in which 100% oxygen has been breathed, the individual has discomfort in the ears and is moderately deaf. Examination of the ear shows that the tympanic membrane is drawn into the middle ear and that the middle ear contains fluid. The discomfort and deafness can be corrected by performing Frenzel's manoeuvre introducing air into the middle ear. The mechanism underlying the condition is similar to that which produces the lung collapse on exposure to +G_z. Breathing 100% oxygen results in the nitrogen normally present in the middle ear cavity being washed out and replaced by oxygen through the pharyngo-tympanic tube (24). With a low concentration of nitrogen in the middle ear cavity, the blood flowing through the wall of the cavity rapidly absorbs gas from it. The absorption of gas reduces the pressure in the middle ear that draws the tympanic membrane into the cavity causing discomfort and deafness. The reduction in pressure also draws fluid into the cavity. The process of absorption of gas from the middle ear can be slowed and arrested after flight by

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inflating the middle ear with air. The re-introduction of nitrogen into the middle ear must be repeated several times over the 12-18 hours following a flight in which 100% oxygen is breathed if delayed otitic barotrauma is to be avoided. However, if several ascents to altitude (even to only 5,000 feet) have been performed whilst breathing 100% oxygen, the absence of ventilation of the middle ear that occurs during sleep frequently results in ear discomfort and deafness the following morning.

Although there is no published evidence of any long-term deleterious effects of breathing 100% oxygen in flight upon the middle ear, many air forces consider that it is an undesirable condition. The incidence of delayed otitic barotrauma is reduced by the presence of a minimum concentration of nitrogen in the gas breathed during flight. The concentration of nitrogen required in the inspired gas to reduce the incidence and severity of this condition to negligible levels is between 30% and 40%

It is concluded, therefore, that in order to prevent the occurrence of acceleration-induced atelectasis and delayed otitic barotrauma in an agile combat aircraft, the maximum concentration of oxygen in the inspired gas should not exceed 60%. There are obvious limits to the altitude range over which this limit can be applied since it conflicts at the higher altitudes with the requirement to prevent hypoxia, which is of paramount importance. Three factors influence the range of cabin altitudes over which the maximum concentration of oxygen should be limited to 60%. The first factor is the pressurisation schedule of the cabin of the aircraft. The aircrew will only be exposed to cabin altitudes greater than 20,000-22,500 feet in the rare event of a decompression of the cabin. The second factor is the effect of altitude upon the ability of an aircraft to sustain high levels of acceleration. Many combat aircraft cannot maintain $+G_z$ accelerations greater than 3-4G at aircraft altitudes greater than 36,000-40,000 feet. Furthermore, air combat is usually performed at aircraft altitudes well below 40,000 feet. Thus the $+G_z$ accelerations required to produce acceleration atelectasis are unlikely to occur at aircraft altitudes above 36,000-40,000 feet, i.e., at cabin altitudes greater than 15,000 feet. The third factor is that it is technically difficult and expensive to control the concentration of oxygen in the inspired gas at a given cabin altitude within the very narrow limits that would be the case if the oxygen concentration was not allowed to rise above 60% at cabin altitudes greater than about 15,000 feet. Thus the minimum concentration of oxygen required at a cabin altitude of 18,000 feet in order to prevent hypoxia on a subsequent rapid decompression is 49% (Figure 1). Consideration of all these factors led to the conclusion that in a typical agile combat aircraft, the maximum concentration of oxygen in the inspired gas should not exceed 60% at cabin altitudes between ground level and 15,000 feet, and that higher concentrations of oxygen are acceptable at higher cabin altitude with the limit that the concentration should not exceed 75% at a cabin altitude of 20,000 feet. These requirements are embodied in current international military standards (Air Std 61/101/6A and STANAG 3865). A typical specification for the maximum concentration of oxygen in a current combat aircraft is presented in Figure 2.

A review of the basis for the requirement that the oxygen concentration shall not exceed 60% at cabin altitudes up to 15,000 feet emphasises that no studies of acceleration-induced atelectasis have been conducted at reduced environmental pressure on a man-carrying centrifuge. The present limit at altitude is based upon the study in dogs of the effects of reduction of environmental pressure on the rate of absorption of gas from non-ventilated alveoli conduced by Ernsting in 1965 (6). It would be valuable if the predictions made on the basis of these animal experiments could be confirmed by studies on man at reduced environmental pressures between ground level and 18,000 feet, especially as the animal studies and calculations suggest that the concentration of nitrogen required to prevent acceleration-induced atelectasis may fall slightly with an ascent to altitude. It has been suggested that the technique of pressure breathing during exposure to $+G_z$ accelerations (PBG) that is being introduced into combat aircraft will prevent the occurrence of acceleration atelectasis even when 100% oxygen is breathed. In-flight assessments of PBG systems with or without chest counterpressure conducted by the RAF Institute of Aviation Medicine do not support this suggestion. The symptoms of acceleration atelectasis have been reported repeatedly by pilots using PBG systems that deliver 100% oxygen throughout flight. This finding is not surprising since lung volumes are not increased by pressure breathing during exposure to $+G_z$ accelerations when wearing G trousers (Green--personal communication) so that PBG would not be expected to prevent closure of the small airways in the lower parts of the lungs during exposure to $+G_z$ accelerations.
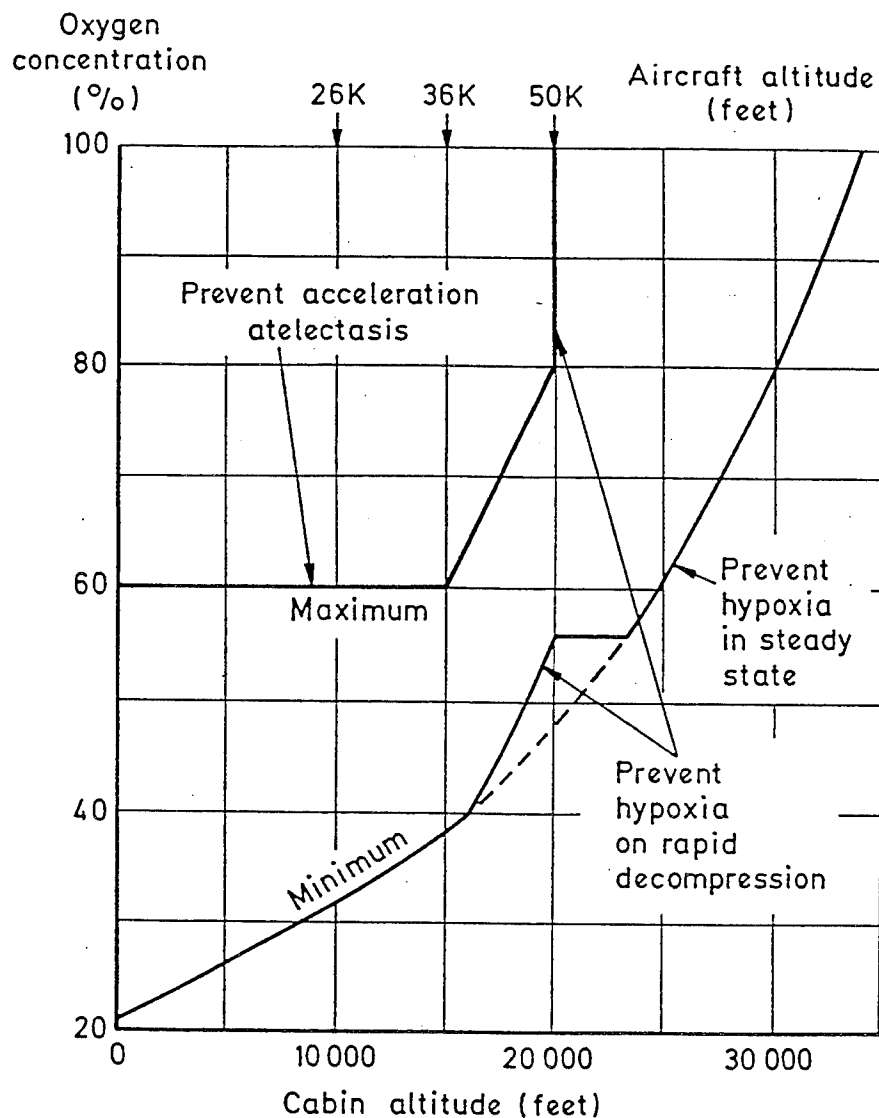
Figure 2. The specification of the relationships between the minimum and maximum concentrations of oxygen in the inspired gas and cabin altitude with the cabin pressurised for a typical agile combat aircraft with a ceiling of 50,000 feet.

Conclusions

The physiological and operational bases of the current international military standards defining the relationships between the minimum and maximum concentrations of oxygen in the inspired gas and cabin altitude in agile combat aircraft when the cabin is pressurised (1, 23) have been reviewed in the light of the results of recent research and the likelihood of such aircraft operating at altitude above 50,000 feet.

This review has confirmed that the minimum concentration of oxygen to be breathed when the cabin is pressurised must be such as to maintain the alveolar PO_2 at, or greater than, 103 mm Hg and to prevent the alveolar PO_2 falling below 30 mm Hg on a rapid decompression of the cabin. The effects of employing pressure breathing systems that will provide protection at altitudes above 50,000 feet upon the latter requirement have been explored for a system that provides a breathing pressure of 70 mm Hg at 60,000 feet. This analysis has demonstrated that the lower the absolute mask pressure provided by such a system, the higher will be the concentration of oxygen that must be breathed before a rapid decompression in order to prevent the decompression reducing the alveolar PO_2 below the acceptable transient minimum value of 30 mm Hg. It has also emphasised that the minimum concentration of oxygen required to meet this requirement varies significantly with the pressurisation schedule of the pressure cabin and the relationship between breathing pressure and altitude provided by the pressure breathing system. Recent experimental studies (16) have re-emphasised the importance of the immediate delivery of 100% oxygen (or product gas containing at least 94% oxygen) to the mask on a rapid decompression to an altitude above 30,000 feet. These studies have reconfirmed the present standards (1, 23) that require that concentration of oxygen in the inspired gas rises to 100% (94% if product gas is being breathed after the loss of cabin pressure) before more than 0.6 litre (ATPD) of gas has been inspired during and after a rapid decompression.

It is concluded that the requirements of Air Standard 61/101/6A(1) and of STANAG 3865 (23) with respect to the minimum acceptable concentration of oxygen to be provided in the inspired gas when the pressure cabin is intact are correct and should be met by aircrew breathing systems to be installed in agile combat aircraft operating above 50,000 feet, as well as those with operational ceiling of 50,000 feet and below.

It has been suggested recently (27) that aircrew operating agile combat aircraft at high altitudes, especially when the cabin altitude during flight will exceed 18,000-20,000 feet, should breathe nitrogen-free gas throughout flight in order to reduce the possibility of severe venous gas emboli or of overt decompression sickness occurring during pressurised flight or following decompression of the cabin. This suggestion has not been adopted by operational air forces except for special tasks, although the US Navy has employed only 100% oxygen in its combat aircraft for many years. The provision of 100% oxygen throughout flight has logistic and breathing equipment design implications, as well as the disadvantages of acceleration-induced atelectasis and delayed otitic barotrauma. Should the latter conditions be considered unacceptable by an air force, then the solution to the avoidance of decompression sickness with the cabin pressurised is to increase the differential pressure of the cabin, especially at the higher altitudes (12). It is hoped that the risks of decompression sickness arising following decompression at high altitude and descent to altitudes between 20,000 and 35,000 feet will be quantified in the next few years so that a balanced judgement can be made as to the need for aircrew operating agile aircraft at high altitude to breathe 100% oxygen throughout flight. It should be emphasised, however, that the molecular sieve oxygen concentrator systems of at least two agile combat aircraft now under development have not been designed to provide nitrogen-free breathing gas when the pressure cabin is intact.

The present review has considered in detail the disadvantages of breathing high concentrations of oxygen in flight in an agile combat aircraft, namely acceleration-induced atelectasis and delayed otitic barotrauma. It has confirmed that both these conditions can be avoided by ensuring that the breathing gas contains at least 40% nitrogen (or nitrogen and argon) at cabin altitude up to the altitude at which the aircraft is capable of applying $+G_z$ accelerations above 3G. It is suggested that whilst the requirement of a minimum of 40% nitrogen to prevent acceleration atelectasis at ground level is well established, further experimental studies are required to confirm whether the concentration of nitrogen (and argon) required to prevent this condition at cabin altitudes above

10,000 feet is as high as 40%. Studies are also required to confirm or refute whether pressure breathing with G (PBG) may reduce the incidence and severity of acceleration atelectasis. The Royal Air Force experience, to date, is that PBG does not do so. Finally, further studies are required to investigate the significance of the large right-to-left shunt of blood that occurs in the collapsed lung in relation to a subsequent exposure to low intrapulmonary pressure.

It is concluded that, until the results of further research are available, the standard that the maximum concentration of oxygen in the inspired gas shall not exceed 60% at cabin altitude up to 15,000 feet (and 75% oxygen at a cabin altitude of 20,000 feet) as required by Air Standard 61/101/6A(1) and STANAG 3865 (23) is sound if acceleration-induced atelectasis and delayed otitic barotrauma are deemed to be unacceptable in aircrew operating agile combat aircraft, as is the case in the Royal Air Force.

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Discussion

PROF ERNSTING: A couple of additional comments. There is nice work being done at Patuxent River suggesting that you can get peak respiratory flows in flight up to 250 liters a minute, but we've not yet introduced that data into our requirements. Also, contrary to what we've heard earlier, we are quite concerned with high Gs at high altitudes.

DR. MOON: Professor Ernsting, to what extent does oxygen atelectasis contribute to arterial hypoxemia? You mentioned a 20% shunt. Presumably that will lower significantly the arterial PO2. Have you made any measurements?

PROF. ERNSTING: Yes, in the 1960 timeframe. I would expect it to drop a few mm Hg, but I'd have to work it out. Once you've got the lung collapse it doesn't re-inflate until the pilot undoes his harness and takes a few breaths getting out of the cockpit. That's when you get all the coughing. We conducted field trials where we actually lifted the pilots out of the cockpits after they had flown either on dilution or 100% oxygen and then X-rayed their chests. Those who were breathing 100% oxygen experienced quite gross lung collapse.

DR. MOON: So at higher altitude then, that could be a significant problem in terms of maintenance of adequate arterial oxygenation?

PROF. ERNSTING: Yes. Also, positive pressure breathing during G is often stated to have a counteracting effect. But in practice, we didn't find that in our flight trials and we didn't find it in the centrifuge studies. So we don't see positive pressure breathing as a criteria for reducing the requirement for having at least 40% insoluble gas in the inspired mixture.

DR. ACKLES: Canada has taken a contrasting view on the 100% oxygen and atelectasis problem. Even though I know we've been part of the airmix school for many years, we recently re-evaluated this condition with respect to our new G protection system and have taken the position that there will be no operationally significant problems with G atelectasis, and we are going to a 100% chest mounted regulator in our new system. We've had no problem with it so far and everyone has accepted this approach. U.S. Navy pilots have experienced the coughing and chest pain associated with G induced atelectasis, but they have not seen any operational problems with breathing 100% oxygen. We haven't shown any kinds of performance decrement, so we have accepted 100% oxygen as our breathing gas, which may make things easier on the pilot if he is exposed to high altitude.

DR. PILMANIS: The next three talks are on decompression sickness. All three speakers will be addressing a common methodology. To save time, I will quickly review the methodology because some of you may not be familiar with the procedures. As many of you are aware, all of our research in DCS at the Armstrong Laboratory goes into a very large computerized database, and many of the things that we'll be addressing in the next couple of talks come directly out of that database. We use a Hewlett Packard SONUS 1000 Echo Imaging System, both here and at Farnborough for bubble detection, that both visually and acoustically records circulating decompression bubbles The protocols vary a great deal. Some involve exercise, some do not. We use both male and female subjects. There are different parameters in each study so we are not able to lump all the data together from the 1500 flights that we have in our database. We have to subdivide them into various types of studies, so the numbers of subjects vary for each specific exposure level, i.e., although we may have a large database, we may have only ten subjects in the study of interest and that opens the question of validity. We are well aware of that limitation and will try to point that out.

An inside observer in the chamber uses a chest positioned precordial transducer to collect data. For the higher flights we've recently instituted the use of a robotic arm. It's working extremely well at 35,000 feet, which is one of the protocols we're conducting at present. The end points on all of these studies are symptoms. We do not use bubble detection as an end point, except if we see gas bubbles cross over to the left ventricle. Then we bring the subject down immediately. We have seen that occur in six subjects to date. We now have a videotape of circulating bubbles in the heart taken by 2-D echocardiography.

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Altitude Decompression Sickness: Operational Significance

James T. Webb, Ph.D.
Andrew A. Pilmanis, Ph.D.

Abstract

Raising the ceiling of current flight operations will have the effect of increasing the altitude exposure hazard and consequent incidence of decompression sickness (DCS) symptoms. In many cases, the current operational incidence of DCS is already a limiting factor, and without increased protection that factor may become the controlling influence for operational planning of some mission scenarios. The F-22 will place the pilot at a cockpit cruise altitude of 22,500 ft, above the threshold of DCS with a latency for symptom onset within one hour (Webb and Pilmanis, 1995c). Use of 100% oxygen is necessary to provide additional protection, and increased cabin pressure differential to at least 6 psid is highly recommended. Research is needed to further define the risk, predict the risk, and offer recommendations for avoidance of DCS symptoms.

Introduction

Decompression sickness currently occurs during some routine training and operational activities (Bendrick et al., 1996; Pilmanis, 1992). That risk will increase if the ceiling of current operations is raised. Plans to raise the ceiling based on increased capabilities of aircraft in development must include consideration of the effect of increased exposure on the pilots, crew, and occupants. For instance, the F-22 will be able to cruise at 60,000 ft. This is a much higher cruise altitude than the operationally realistic cruise altitude for current fighters of about 35,000 ft. The higher altitude is to be maintained with the same cockpit differential pressure, 5 psid, designed into fighters of the 50s and 60s.

Objectives

The questions posed in the introduction have provided Armstrong Laboratory (AL) High Altitude Research personnel with specific program objectives. One objective is to quantify the risk associated with various operational activities. Another is to develop risk prediction methods to quantify that risk in such a way that the methods could be used by mission planners. Quantification and prediction of risk permit accomplishment of the third objective, to recommend ways the risk can be minimized or eliminated.

The purpose of this paper is to provide background data from the AL research program as it relates to accomplishment of operational missions. Some of the findings have led to recommendations that improve denitrogenation efficiency by reducing the time spent preoxygenating at ground level prior to take off (Pilmanis and Olson, 1991). Some of the findings have also prompted additional research to isolate the altitude threshold of DCS without denitrogenation and to determine the true relationship between denitrogenation time and incidence of symptoms.

Pilots Use of "Normal" vs 100% Oxygen

The USAF Narrow Panel Regulator delivers up to 50% nitrogen at altitudes between about 20,000 ft and 24,000 ft with the Diluter Lever in the **NORMAL** position. Mid-1980s AL chamber research results using a breathing mixture of 50% oxygen and 50% nitrogen (Webb et al., 1990) are, therefore, applicable to operational

scenarios in which the cockpit altitude is between 20,000 ft and 24,000 ft and the pilot has **NORMAL** selected. Although the pilot will not become hypoxic at these altitudes with up to 50% nitrogen in the breathing mixture, little denitrogenation occurs during flight. The research on the relationship between breathing gas and incidence of venous gas emboli (VGE) concluded that breathing 100% oxygen is significantly more effective at preventing VGE below 20,000 ft than breathing a mixture containing 50% nitrogen and 50% oxygen (Webb and Pilmanis, 1993). Since 100% oxygen (0% nitrogen) is typically available for fighter aircrew, all recent chamber research at AL has used 100% oxygen as the breathing gas.
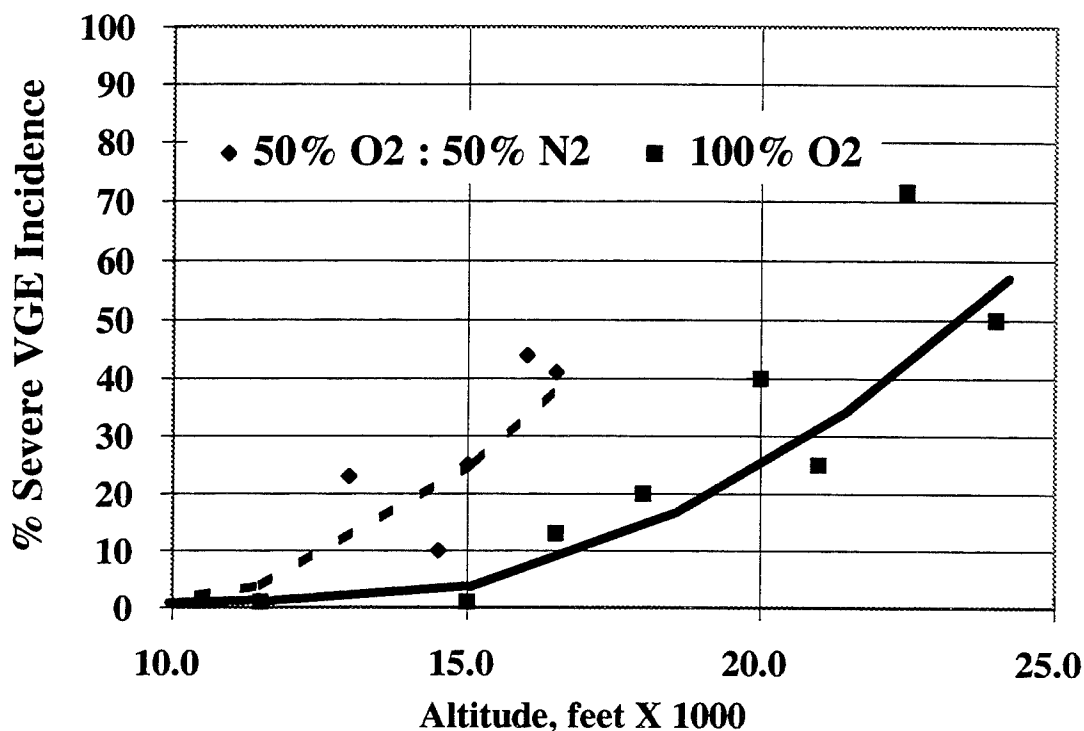
Figure 1. Effects of Breathing 50%N$_2$ and 50%O$_2$ vs 100%O$_2$

Figure 1 shows the incidence of severe VGE (Spencer Scale Grades 3 and 4 VGE) (Webb and Pilmanis, 1993) versus altitude with two breathing gases. Subjects were exposed to the altitudes shown, without preoxygenation, for at least 6 hours while breathing either a 50% nitrogen:50% oxygen mix, or 100% oxygen. Figure 2 shows that even with 100% oxygen as the breathing gas, VGE occur within 30 min at a cockpit altitude of 22,500 ft. These VGE increase the hazard of further decompression, in particular, unplanned rapid decompression (RD) (Webb et al., 1993). The hazard, expansion of preexisting gas emboli during decompression, relates to a designed cruise altitude of 60,000 ft for F-22 and Eurofighter 2000 aircraft. With a 5 psid pressurization system, the pilots of these aircraft will experience 22,500 ft throughout cruise and any unplanned RD late in cruise could lead to very rapid onset of DCS due to physical expansion of the existing VGE and extravascular emboli (Webb and Pilmanis, 1995b). With positive pressure breathing for altitude (PBA), the lung would only experience a decompression to about 42,500 ft, not 60,000 ft. However, a decompression from 22,500 ft to 42,500 ft represents a 2.53-fold (314/124) emboli volume increase or an increase in emboli diameter of about 36%.
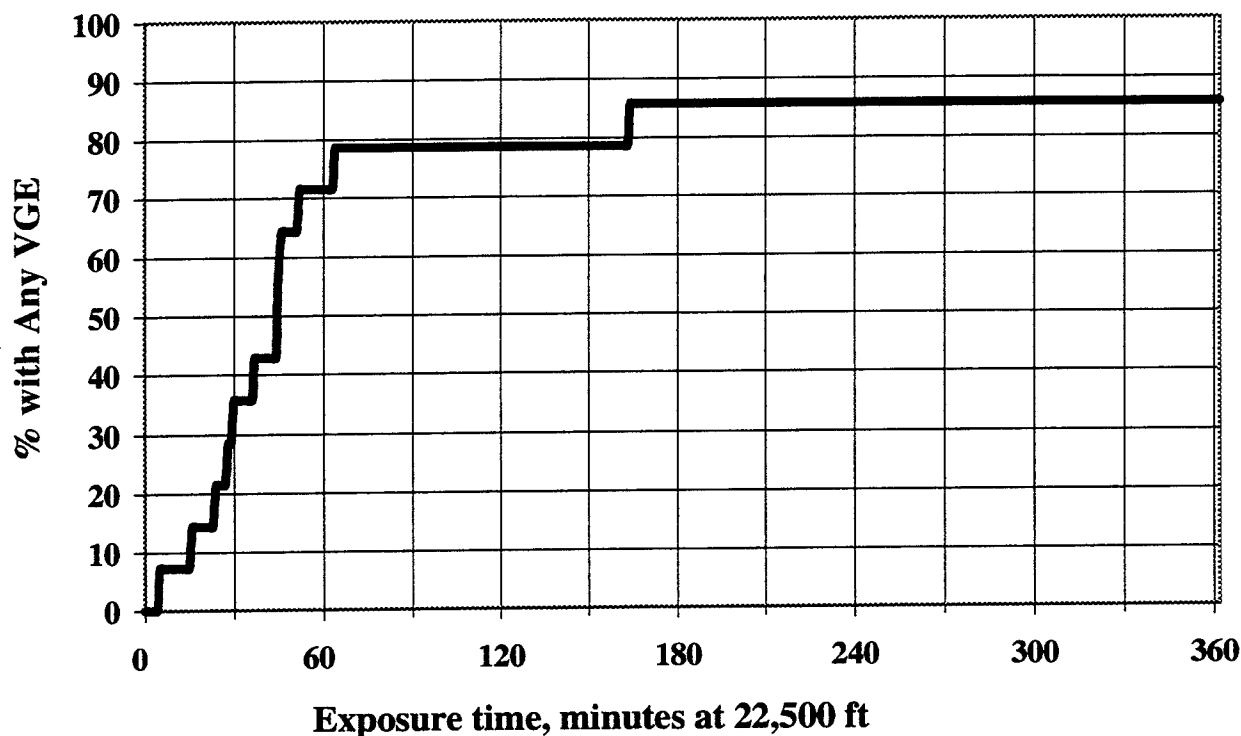
Figure 2. Zero-Preoxygenation VGE at 22,500 ft

Figure 3 shows that the zero-preoxygenation DCS threshold is apparently between 21,200 ft and 22,500 ft. Approximately 10 different subjects were exposed at each altitude shown (75 total). Increasing the N to 20 males and 20 females at 21,200 and 22,500 ft is underway. If the increase in DCS incidence from 21,200 ft to 22,500 ft remains this notable, we will be both surprised and very curious as to why. The operational significance of this range of altitudes is that it coincides with a pilot's environment in a 5-psid cockpit flown at 60,000 ft, for instance, the F-22 and EuroFighter 2000. Although many current fighters can reach this altitude, these new aircraft expand the ceiling of normal cruise, which will place them at approximately 22,500 ft long enough to develop symptoms as shown in Figure 4.
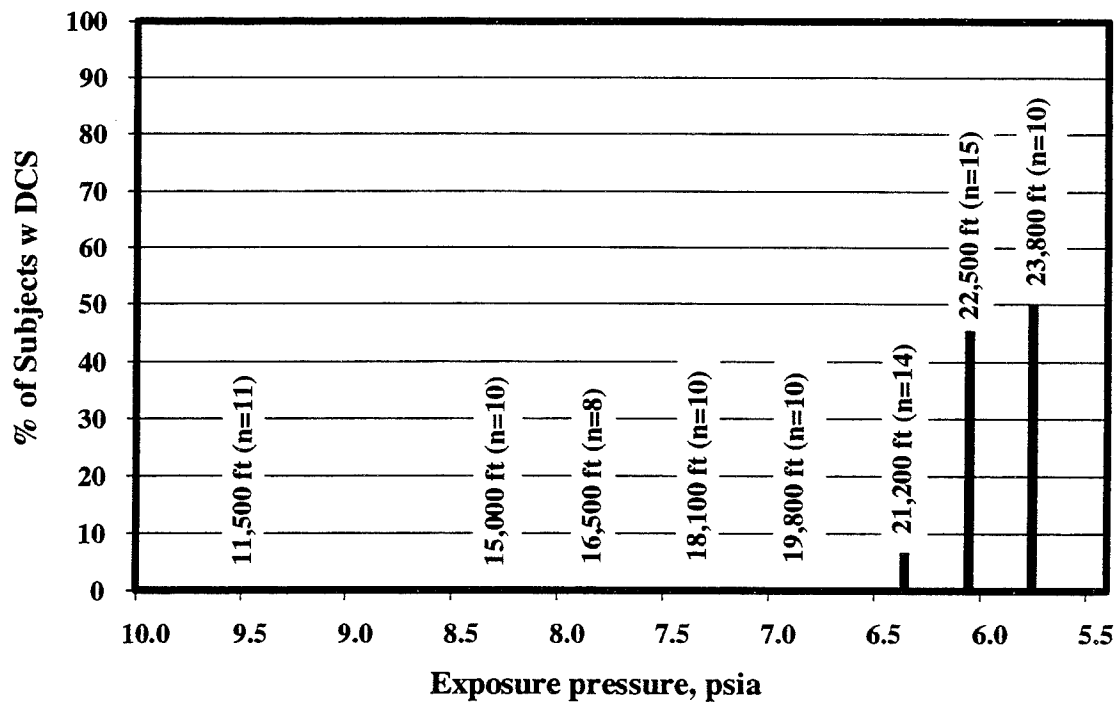
Figure 3. Zero-Preoxygenation DCS Threshold
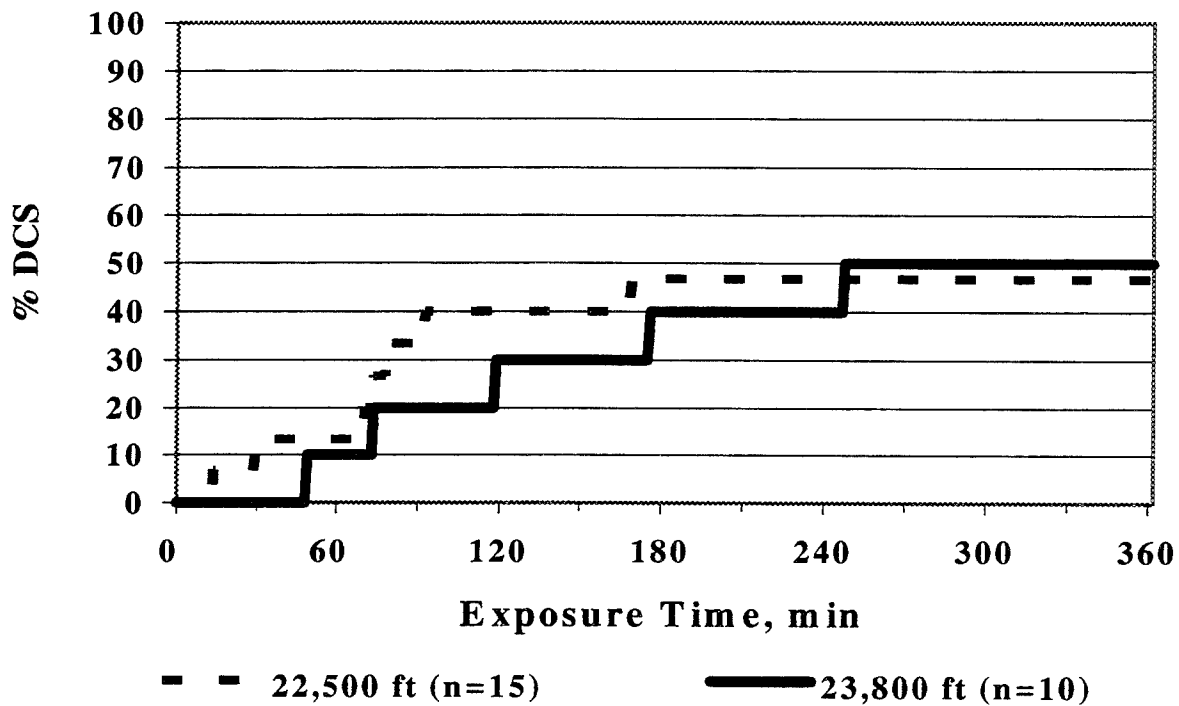
Figure 4. Cumulative DCS Symptom Incidence at 22,500 ft and 23,800 ft

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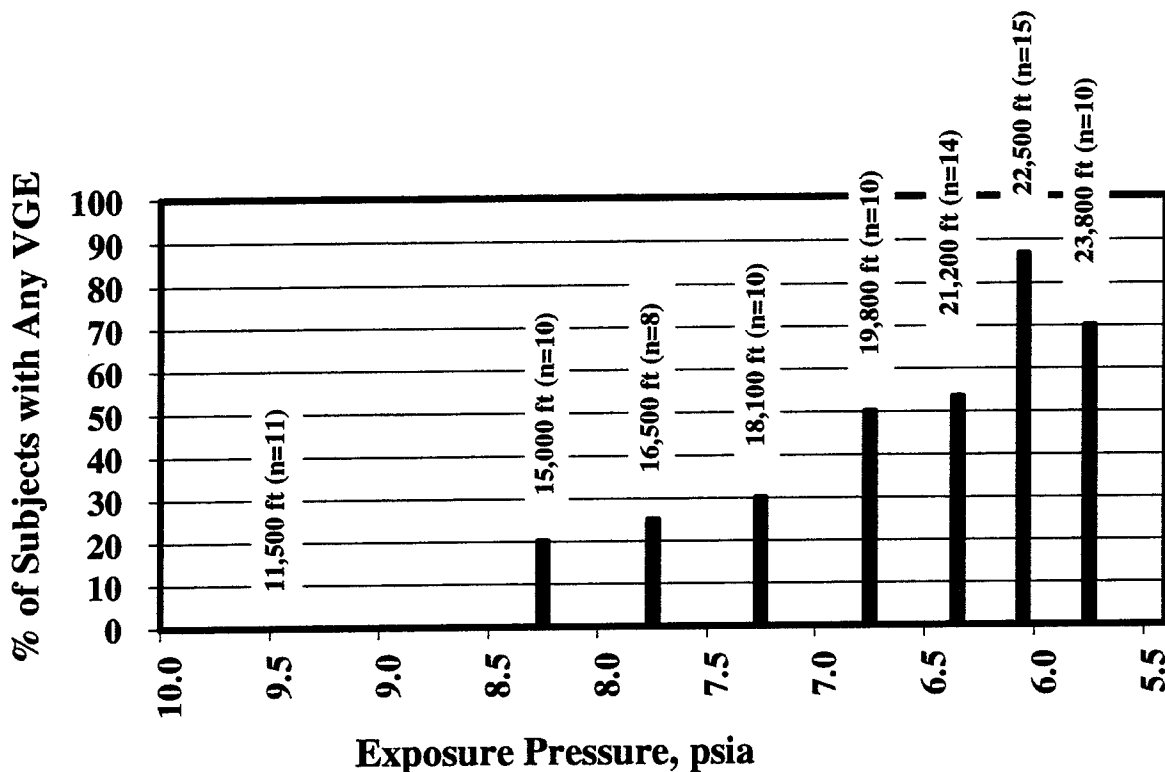
Figure 5. Zero-Preoxygenation VGE Threshold

Although 18,000 ft has been used as the theoretical threshold for DCS, that theory was based on Haldane's hyperbaric work reported in 1908, in which hypobaric DCS was not discussed (Boycott et al., 1908). The 25,000 ft threshold emanating from operational, conventional wisdom is probably based mostly on a sparse number of operational reports indicating that inability to perform the mission due to DCS was rare below 25,000 ft.

If a well-defined threshold can be identified experimentally, it could provide insight into the risk associated with F-22 operations (as well as risk in other aircraft, such as the T-37) where the cockpit altitude can exceed 21,000 ft. In addition to the DCS hazard inherent with 1-3 hour residence times at an altitude of 22,500 ft, an F-22 pilot could face the additional risk of further decompression to 60,000 ft with rapid growth of any gas emboli existing at the time of decompression.

Development of VGE in over 70% of subjects exposed to at least 22,500 ft (see Fig. 5) indicates that further decompression could result in rapid enlargement of the preexisting gas emboli in the vasculature or in tissues. The rapid emboli enlargement could result in symptom development with very little latency in comparison to the latency for symptoms when no preexisting emboli are present. Several preventive measures could reduce the hazard of DCS symptom development.
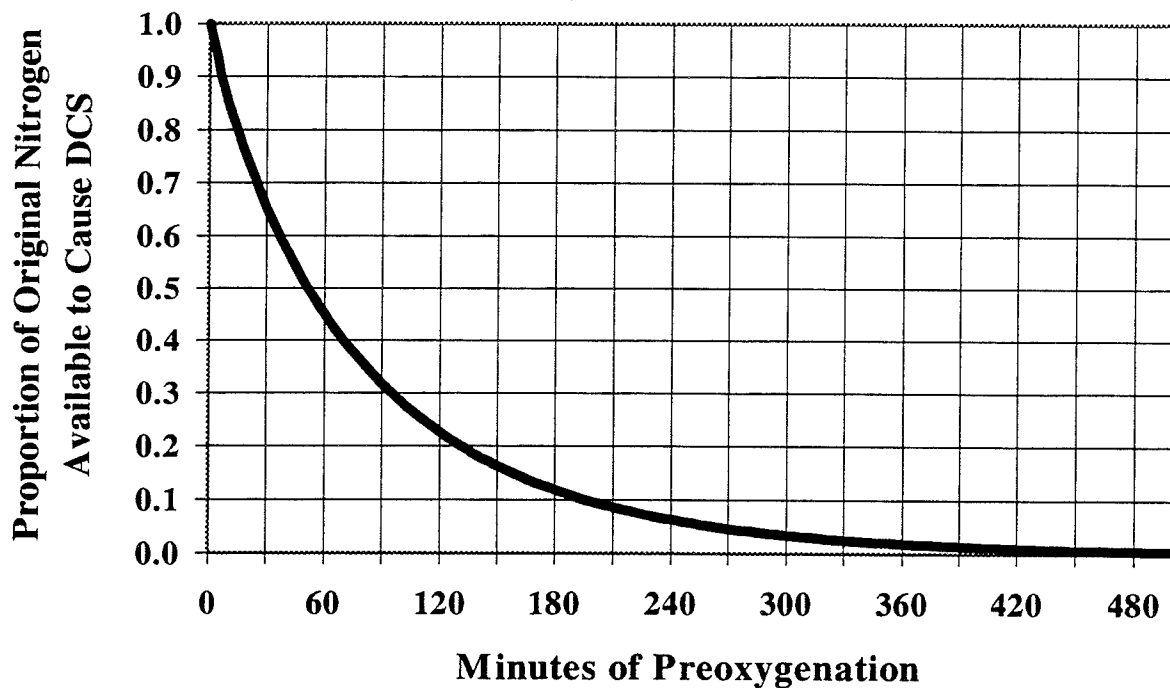
Figure 6. Preoxygenation time vs proportion of nitrogen available to cause DCS

Preoxygenation (prebreathing) reduces the body's store of nitrogen, thereby reducing the potential for development of gas emboli and DCS symptoms. Increasing preoxygenation time results in more denitrogenation which, in turn, reduces level of DCS incidence for any given exposure. That relationship is probably not linear. The shape of the curve in Figure 6 is modeled after the shape of a curve plotting nitrogen elimination versus time. What is not known is the relationship between amount of nitrogen eliminated and its affect on DCS incidence. The first nitrogen to be eliminated is the nitrogen in the lung and blood, which has little or no effect on DCS incidence. Therefore, since this first bolus of nitrogen represents a large percentage of nitrogen in the body, it can lead to misinterpretation of the value of its removal. Thus, the shape of a curve relating DCS incidence to preoxygenation time may be somewhat flat at the top for the first hour, followed by a sharp decrease in DCS incidence when the slow tissues begin to denitrogenate, then leveling as the incidence of DCS drops toward zero. The duration of preoxygenation required will depend on the altitude, duration of exposure, and activity while decompressed. These factors make it is very difficult for operational planners to develop estimates of DCS incidence.

The frequent inquiries about DCS risk received at the Armstrong Laboratory resulted in the preliminary development of tables that could be used in mission planning until an effective decompression computer is developed and transitioned to the operational community (Tables 1 & 2). In this development, we stipulated use of 100% oxygen as the breathing gas during the exposures because 0% nitrogen breathing gas (molecular sieve oxygen generated; MSOG) is planned to be available in advanced fighter aircraft and because it is recommended for use during high-altitude flight as described earlier. Preoxygenation times were based on experimental data and current operational procedures.

Among the interdependent conceptual relationships that must be considered when deriving table values are DCS incidence versus exposure time, altitude, and exercise. A review of these basic conceptual relationships will set the stage for showing how these tables were developed (Webb and Pilmanis, 1995a).

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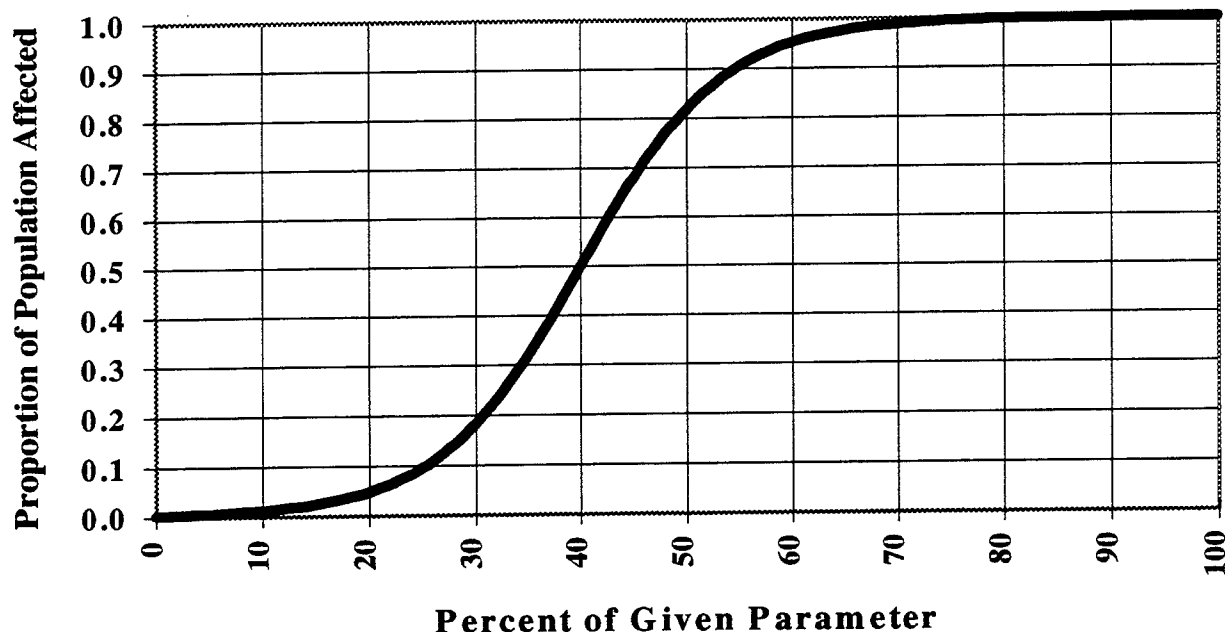
Figure 7. Sigmoidal Relationship between DCS Incidence and Variable Parameter

The effects of exposure time, altitude, and exercise follow a basic sigmoidal curve such as the one shown in Fig. 7, in which the parameter has little effect at very low levels. At some higher level of a parameter, the incidence of DCS rises rapidly followed by a leveling at the upper limits of the effect. This leveling of effect may occur well below 100% incidence, depending on the effects of other parameters involved.

Tables 1 and 2, if used in accordance with the limitations described in Webb and Pilmanis (1995a), should provide a reasonable guide until a better method is available, namely, the DCS computer model under development at the Armstrong Laboratory.

One of the objectives of the AL High Altitude Research program is to provide recommendations emanating from research that may be of value to the operational community. Results of recent research indicate that denitrogenation (preoxygenation) accomplished at altitudes up to 16,000 ft is as effective as denitrogenation at ground level. This effect was not true at 18,000 ft. The difference between 16,000 ft and 18,000 ft can be seen in the severe VGE response shown earlier (Fig. 1). These findings could be of use to F-22 pilots if enroute altitudes could be maintained below 38,000 ft (16,000 ft cockpit altitude) until further climb to 60,000 ft was necessary.

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Table 1. ESTIMATED DCS RISK AT 22,500 AND 25,000 FEET

Exposure Duration, h	Physical Activity[1]	Preoxygenation Time, min	Estimated % DCS[2]	
			22,500 ft	25,000 ft
4	Heavy Exercise	0	80	95
		60	55	85
		90	40	80
	Rest	0	50	90
		60	35	75
		90	30	65
3	Heavy Exercise	0	75	90
		60	50	80
		90	35	75
	Rest	0	50	85
		60	30	70
		90	25	60
2	Heavy Exercise	0	65	80
		60	40	60
		90	25	50
	Rest	0	40	65
		60	15	45
		90	10	35
1	Heavy Exercise	0	25	35
		60	15	20
		90	10	10
	Rest	0	10	25
		60	5	10
		90	5	5

Table 2. ESTIMATED DCS RISK AT 18,000 AND 20,000 FEET

Exposure Duration, h	Physical Activity	Preoxygenation Time, min	Estimated % DCS[2]	
			18,000 ft	20,000 ft
4	Heavy Exercise[1]	0	25	45
		30	15	20
	Rest[2]	0	<10	<10
		15	0	5
3	Heavy	0	20	40
	Exercise	30	15	20
	Rest	0	<10	<10
		15	0	5
2	Heavy	0	15	30
	Exercise	30	10	15
	Rest	0	<5	<5
1	Heavy	0	5	15
	Exercise	30	5	10
	Rest	0	<5	<5

[1]   Heavy exercise is defined as physical activity exceeding 50% VO_{2peak}; Rest is defined as physical activity not exceeding 20% VO_{2peak}

[2]   The shaded, bold-faced percentage numbers for % DCS in the tables are supported by research chamber results. All other values were estimated from the research chamber results using the concepts described in the text.

Recommendations

Research at AL has provided data in support of several recommendations pertinent to the issue of raising the ceiling of operations. Use of 100% oxygen (or 100% MSOGS product gas; less than 2% nitrogen) in lieu of the breathing mixture delivered by the USAF Narrow Panel Oxygen Regulator in the NORMAL position would provide **significant** additional protection for those crewmembers who will be exposed to altitudes above 16,000 ft, particularly if they experienced a further decompression at a later time.

Inflight denitrogenation with 100% oxygen has been shown to be effective and should be used at or below 16,000 ft cabin altitude.

The altitude DCS prediction tables (Tables 1 & 2) can be used much like the analogous USN diving tables.

Increasing the differential pressure in future fighter aircraft cockpits from 5 psid to 7 psid would be beneficial in reducing DCS. Without this protective measure, one or more of the following consequences must be accepted: 1) DCS symptom development after cruise at 60,000 ft for more than approximately 30 min; 2) increased hazard of rapid-onset symptoms of DCS in the event of unplanned further decompression; 3) requirement for at least 1 h of preoxygenation; and 4) use of a full-pressure suit.

Research

Research will be necessary on the effects of increased cockpit differential pressure prior to design criteria modification. Continuation of research on defining the zero-preoxygenation threshold could allow more practical denitrogenation requirements at the altitudes surrounding the rapid increase in DCS incidence. Research in support of operational problems associated with raising the ceiling is needed in the areas of repeat exposures and effects of moderate exercise at altitude on DCS incidence.

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Discussion

DR. ACKLES: Do you have any evidence that repeated exposures, like daily exposures, reduces the bubbling?

DR. WEBB: We don't have that data yet. A repetitive altitude exposure study is planned later this year. I would like to add a comment with respect to acceleration atelectasis. The Navy uses 100% throughout their flight regime in tactical aircraft and has not reported an operational decrement resulting from acceleration atelectasis. I would suggest that from take off to high cruise and supercruise the pilot use 100% oxygen continuously to reduce the risk of DCS and if he must come down for engagement at lower altitudes, the concentration could be lowered to reduce the effects of atelectasis. Supercruise at high altitude probably does not involve more than 3-4 G and then only for short periods of time. Would you like to comment Maj. Neubeck?

MAJ. NEUBECK: Yes. You're going to lose energy fairly rapidly trying to maintain 3 or 4 Gs at high altitude. You're probably not going to be able to sustain that level of G for very long.

DR. WEBB: By breathing a high concentration of oxygen, you may reduce the DCS problem somewhat and if you must then come down and fight the battle at a higher G level, the oxygen should be switched to normal airmix. If you're going to be engaged, you're going to be breathing deeply with nitrogen in the mixture to help reduce the atelectasis problem.

DR. SEARS: I completed a review on repetitive decompressions for NASA a while back and much of the early data indicated there is no problem for daily altitude exposures. As a matter of interest, one study found less bends after 30 days of daily exposure to high altitude. Other studies found that if exposures were repeated in less than 3-4 hours, a greater incidence of DCS occurred.

Rapid Decompression and Decompression Sickness

Robert W. Weien, Lt Col, USA, MD, MPH

Introduction

The operational environment will demand that future generations of aircraft will operate at higher altitudes. Improved weaponry and opposing force aircraft will require the greater protection afforded by altitudes in excess of the current limits. The physiologic environment becomes ever less forgiving as one goes higher. One aspect of concern is the effect of rapid decompression (RD) on the onset and severity of decompression sickness (DCS).

Work under way at Armstrong Laboratory, and at the Defence Research Agency Centre for Human Sciences at Farnborough is aimed at determining the incidence and severity of decompression sickness in the aviation environment. Flights to 40,000 feet have been accomplished, and may extend higher still.[1] In these studies preoxygenation occurs and the rate of ascent is relatively slow. Thus, an unanswered question is the effect of rapid decompression: to what extent does RD affect the onset and severity of DCS, and to what altitude must a fighter descend in order to minimize its effects should an RD occur?

This paper will briefly review the literature concerning rate of ascent on DCS, and then present work both underway and planned at Farnborough.

Literature Review

One of the few studies using human subjects to investigate this is also one of the earliest. Hitchcock, Whitehorn and Edelmann reported in 1948 on a series of human subject exposures.[2] They conducted a series of five studies comparing rates of ascent of 0.7 psi per minute (the slow rate), to 0.7 psi per second or greater (explosive decompression). The highest rate used was 4 psi/second.

The findings were mixed. Of the five studies, two showed that the RD group had a greater rate of DCS than did the control, whereas three showed no statistical difference. The profiles and results are summarized in Table 1.

Table 1: Hitchcock results summary.

Groups*	Statistical Difference	Oxygen use	Exercise	RD altitudes (1000s ft)	Final altitude and time
I	Yes	100%/ascent	Rest	20-40	35K/60'
II/III	No	100%/ascent	Exercise	20-40	38K/90'
IV	Yes	100%/ascent	Exercise	10-35	38K/90'
V	No	Airmix	Exercise	10-35	38K/90'
VI	No	100%/ascent	Rest	27.5-40	45K/60'

* Hitchcock's group numbering retained. Groups II and III followed the same profile, but Group II had male subjects, and Group III had female subjects.

Hitchcock's conclusion was that "...explosive decompression at the rates and ranges used in these experiments produces a slight increase in susceptibility to decompression sickness." In his opinion the effect of RD is slight, and masked by other, more significant factors, thus explaining the non-significant results in three of his

studies. He also concluded that "...explosive decompression, within the rates and ranges used in these experiments, does not constitute a serious hazard to normal human beings."

Fryer in 1969[3] found that there was little basis for drawing firm conclusions about the effect of RD on DCS. He concludes that "long and exhaustive series of experiments on groups of men would be required to investigate fully the effects of varying rates of ascent within the practical aviation range." His final opinion, however, was that "on practice, it is generally recognized that wide variation in ascent are without great effect on outcome."

Piwinski, et. al, in 1986[4] reviewed more than five years of experience at one training hypobaric chamber. They concluded that RD did not increase the incidence of DCS. The type of RD profiles used in this chamber are identical to those of the USAF.

Kumar and Walligora in 1989[5] reported that their subjects divided into two categories: the rates differed significantly in those in which the rate of ascent was <2500 feet/minute, and those >2500 feet/minute. The higher rate group included exposures up to 53,000 feet/minute. Within the two bands there were no significant differences.

Baumgartner and Weien in 1992[6] noted that the USAF Type II altitude chamber profile had the highest incidence of DCS. This profile includes an RD (8,000 feet to 22,000 feet) but it also has the highest altitude of routine USAF training flights (43,000 feet). Thus, the effect of the RD may be masked by the effect of the higher altitude.

That the effect of RD on DCS incidence is a valid operational concern was highlighted by Brooks in 1984[7]. He reported on 47 incidents of loss of cabin pressure over a 20 year period. The final altitude of these cases ranged from 15,000 to 54,000 feet. There were 2 cases of DCS in this population.

Current CHS Study

A project recently approved at the CHS is designed to address this problem. The risk to mission completion represented by rapid decompression in the current Nimrod or future Replacement Maritime Patrol Aircraft is not known. It is well understood that hypoxia can be prevented by the standard breathing systems on board those aircraft, but the risk of DCS is unknown.

The aim of this study is to evaluate the risk of DCS and venous gas emboli (VGE) following loss of cabin pressure and subsequent sustained altitude exposure as required by current mission profiles. Those procedures allow for mission at a cabin altitude of 25,000 feet. This study simulates an RD early in the mission profile, with continuation of the mission at 25,000 feet.

There will be 15 subjects completing each of 3 profiles. The profiles are outlined in Table 2, and presented graphically in Figure 1.

The changes between profiles are designed to examine different aspects of the DCS risk factors. In profile 1, the major change from previous 25,000 foot exposures at Farnborough is that it will be zero prebreathe. Profile 2 adds the effect of an RD prior to the sustained exposure at 25,000 feet, and profile 3 changes the breathing mixture to airmix. This final set of variables most closely simulates the operational environment targeted by the study. The air-oxygen breathing mixture used in profile 3 will be representative of that provided by a Type 417 Mk 2 regulator, and will maintain an alveolar oxygen tension of not less than 100 mm Hg at 25,000 feet.

The profiles are designed to lead gradually from the lowest perceived DCS risk to the highest. Thus, a minimum of 6 subject exposures will complete profile 1 before profile 2 is begun. Likewise, at least 6 subjects will complete profile 2 before profile 3 exposures begin.

Every 15 minutes while at 25,000 feet, the cardiac chambers will be monitored using 2D and Doppler ultrasound with a commercial clinical ultrasound machine (Hewlett-Packard SONOS 1500). An inside observer, or robot arm controlled by an investigator outside the chamber, will position and manipulate the ultrasound transducer on the subjects' chest in a modified long axis view. The subject will move each limb in sequence, so as to dislodge VGE, and these will be visualized as they pass through the right heart.

Table 2. 40K RD profiles.

	Profile 1	Profile 2	Profile 3
Prebreathe	Zero	Zero	Zero
Breathing Mix	100% from ascent	100% from ascent	Airmix, except RD
RD	None	8K to 40K	8K to 40K
Final Altitude	25K	25K	25K
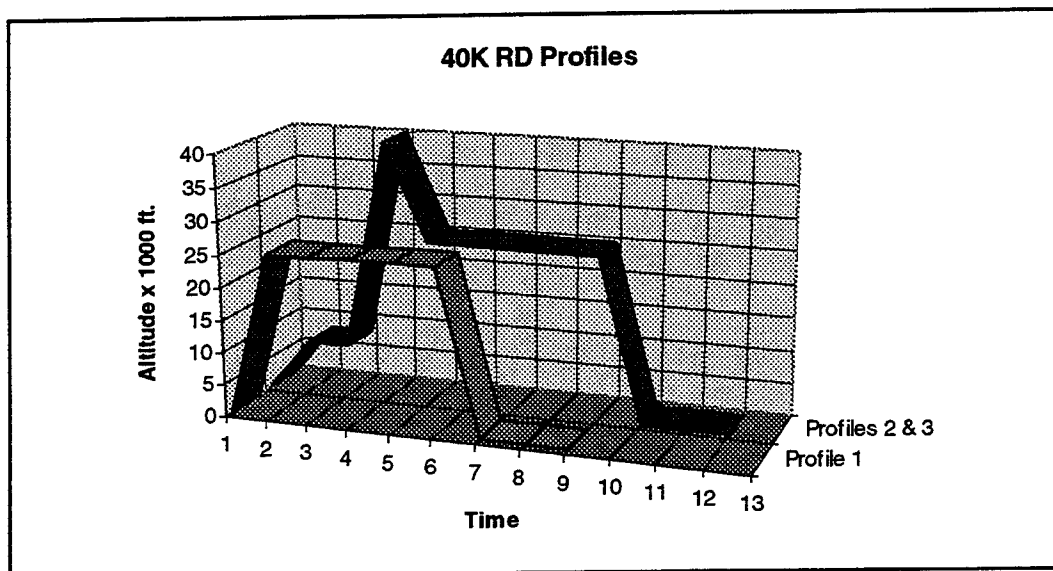
Figure 1: 40K RD Profiles.

Other variables monitored during the exposure include ECG, chamber altitude, oxygen saturation (via pulse oximetry, during RD only), and end tidal PN_2, PO_2, and PCO_2 (via mass spectrometry).

Endpoints for this study are well defined. If any of the following should occur to either the subject or the IO, both will be brought back to ground level, on 100% oxygen (if not already breathing 100%, they will switch to 100% oxygen):

a) the subject or the IO experience joint pains which are mild to moderate, or more severe, and continuous.

b) the subject or IO experience any other symptoms that may be related to DCS.

c) VGE are detected in the left cardiac chambers

d) subject or IO request descent.

Post exposure, the subject will breathe 100% oxygen for two hours, whether or not symptoms or VGE developed. If symptoms persist to ground level, or were more severe than joint pains, a recompression treatment will be conducted in a hyperbaric chamber.

Future CHS Study

A protocol under development at CHS is to investigate the effect of RD on DCS incidence at higher altitudes, up to 60,000 feet. This study would support the EF2000 program, and be applicable to the F-22 program.

In this study, like the 40,000 foot RD study, a cautious and stepwise progression toward the anticipated operational profile is planned. All flights in this study will be zero prebreathe. The profiles for this proposed study are illustrated in Figure 2. They are:

a) 35,000 feet for 60 minutes, on 100% oxygen.
b) RD from 22,500 to 60,000, then 60 minutes at 25,000 feet, on 100% oxygen.
c) RD from 22,500 to 60,000, then 60 minutes at 35,000 feet, on 100% oxygen.
d) 1 hour at 22,500 on 100% oxygen, then RD to 60,000, then 60 minutes at 35,000 feet, on 100% oxygen.
e) 1 hour at 22,500 on airmix, then RD to 60,000, then 60 minutes at 35,000 feet, on 100% oxygen.
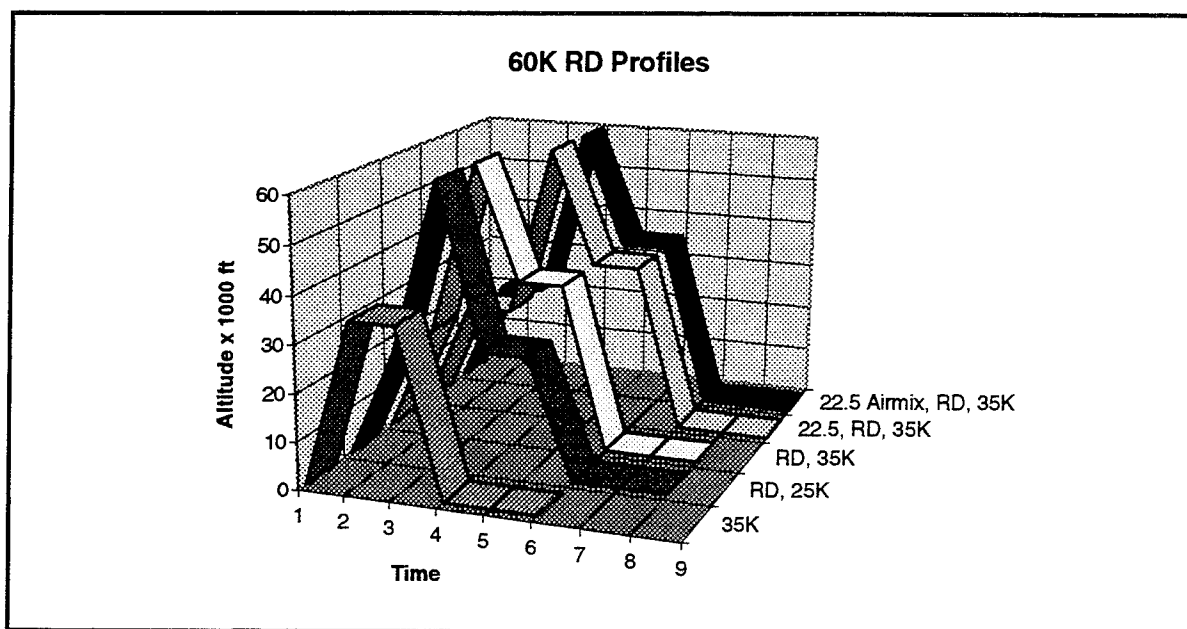
Figure 2. 60K RD Profiles.

These profiles are expected to be more provocative for DCS than those in the 40K DCS study, and will thus be approached with more caution.

Conclusions

The effect of RD on the incidence, onset, and severity of DCS and VGE is not well understood. Previous research in the field is largely absent, and the studies that are to be found are inconclusive. In addition, no work has been performed to establish the effect of RD at altitudes at and above 40,000 feet.

A study of the effect of RD on DCS is important, especially as operational demands force the military aviator to higher altitudes.

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Discussion

MAJ. KREBS: How do you establish the time you're going to use as a rapid decompression?

LT. COL. WEIEN: A lot of that is being driven by chamber capability. I think we decided that the decompression should occur in no more than 3 seconds.

LT. COL. DEMITRY: Is there a model that would predict the level of DCS during each protocol?

LT. COL. WEIEN: No. There is no model that I'm aware of. Is anybody else aware of?

DR. PILMANIS: Not yet.

LT. COL. VANDERBEEK: Are any of your studies looking at less than 100% prebreathe, i.e., OBOGS product gas? Any other studies planned for 93-94% oxygen prebreathe?

LT. COL. WEIEN: No.

Decompression Hazards
at Very High Altitudes

Andrew A. Pilmanis, Ph.D.

Abstract

The use of high-altitude air space for military activities exposes flight crews to the hazards of near vacuum ambient pressures. Advanced fighter aircraft, such as the F-22 and Eurofighter 2000, will have the capability to sustain normal cruise flight at an altitude of 60,000 ft or higher. Information about decompression sickness occurring at or above altitudes of 40,000 feet is minimal. Of critical concern is the flight scenario in which 1-2 hours of cruise with the pilot at a cabin altitude of 22,500 ft is followed by an unplanned decompression to the ambient altitude of 60,000 ft. In this scenario, existing gas emboli will rapidly expand, resulting in potentially serious symptoms with short onset times. This problem could further be exacerbated if descent to low altitude was not immediately possible.

Arterial gas emboli are generally viewed with great concern. Previously unreported left ventricular gas emboli were observed with echo imaging in six volunteer subjects during exposure to simulated altitude. In all 6 cases, at the time of arterial gas emboli onset, the venous gas emboli scores were high from all monitored sites. It was concluded that this gas transferred from the venous side to the arterial side via either intracardiac defects, pulmonary shunts, or pulmonary microcirculation. It is suggested that operational altitude exposures known to elicit high VGE counts in the majority of people should be avoided because of an increased risk of right-to-left gas cross-over and the resulting potential of severe cerebral symptomatology.

Ebullism, or the vaporization of body fluids, poses additional physiological risks to flight above 63,000 feet. Medical treatment protocols for ebullism in the event of accidental manned exposures to extreme altitudes do not exist.

Introduction

Ascent to altitude can lead to the development of clinical symptoms and pathological changes collectively known as decompression sickness (DCS), and to the vaporization of body fluids, a condition called ebullism (32,33). DCS occurs as a result of the evolution of nitrogen to form bubbles in the tissues. This inert gas bubble formation occurs when ambient pressure falls low enough and/or rapidly enough for gas phase separation to occur. These gas bubbles can obstruct circulation, disrupt tissues by distention, and alter biochemical and hematological balances, resulting in a complex myriad of clinical manifestations ranging from local joint pain, to neurological effects, to complete circulatory collapse and death (15,28).

In any high-altitude operation, the inherent risk of rapid decompression must be considered. Conventional protection against DCS and ebullism include prebreathing, cabin pressurization, full-pressure suits, and , in some cases, partial-pressure suits. Measures to deal with the failure of these systems must be defined. In high-altitude reconnaissance aircraft such as the U-2, the risk of DCS has historically been high because the cabin pressurization system maintains the pilot at 28,000 to 30,000 ft (4, 25, 40). Currently, full-pressure suits are used in these aircraft in case of loss of pressurization. These suits are not generally considered practical in fighter aircraft because of restricted mobility, poor comfort, and logistical problems. For these aircraft, partial-pressure ensembles (with no helmet) that provide both G-protection and hypoxia protection are being implemented. These garments only provide partial protection against the severe physiological consequences of DCS and ebullism. Human experiments at very high altitudes have demonstrated that conscious survival for very short "get-me-down" scenarios is possible wearing partial pressure protection with positive pressure breathing. However, the physiological risks of positive

pressure breathing at very high intrapulmonary pressures are complicating factors. In addition, the onset and severity of DCS and the catastrophic effects of ebullism at these very high altitudes also need further elucidation.

Operationally, DCS has been of little concern in previous and current fighter aircraft because these aircraft have been limited to an altitude ceiling of 50,000 ft for relatively short exposure times. However, the next generation of fighter aircraft, such as the F-22 and Eurofighter 2000, may have the capability to sustain normal cruise flight at altitudes up to 60,000 ft. Considering current trends for fewer forward bases and longer flights using inflight refueling, such high-altitude exposure could be many hours in duration.

This paper is limited to a discussion of the conditions of DCS and ebullism associated with exposure to altitudes above Flight Level (FL) 400. The general purpose of the paper is to discuss the current understanding of these two conditions, and to explore critical areas of physiological research necessary for expanded flight operations to higher altitudes. Figure 1 illustrates the potential scenarios and options associated with raising of the operational ceiling from 50,000 ft to 60,000 ft.
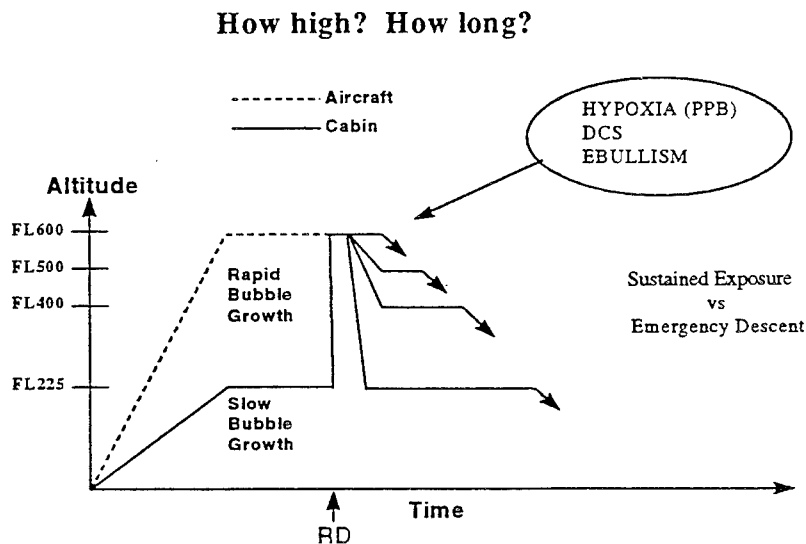
Figure 1. Loss of pressurization options.

DCS and Bubbles

It is well known that DCS severity increases and onset times decrease with altitude (15). Furthermore, rapid decompression to altitudes above 40,000 feet carries higher risk than slow ascents or ascents to lower altitudes. However, few data are available on DCS in humans at altitudes above 40,000 ft. In-vitro bubble growth experiments have shown that, at relatively low altitudes, i.e., less than 25,000 feet, bubbles grow at a gradually decreasing rate and reach relatively small diameters. At high altitudes, i.e., over 30,000 feet, the bubbles maintain a period of rapid growth before tapering off, and attain proportionately larger diameters (29). Asymptomatic intravenous bubbles have been detected as low as 10,250 feet (10), while at higher altitudes, venous gas emboli (VGE) and symptoms frequently occur in close succession (24). Olson and Krutz (29) concluded that the rate of bubble growth and the resulting bubble size is the critical factor in the latency and development of DCS. Petropoulos (31) modeled bubble dynamics and found that the bubble size was dependent on diffusivity, bubble density, bubble nucleation rates, and altitude. He found that bubble diameter growth for rapid decompressions from 22,500 to 60,000 ft was very rapid, i. e., approximately 2 to 3 seconds to grow from 180 to 340 microns.

The onset of symptoms is probably dictated not only by the size of the bubbles, but also by the number and location of these bubbles. Bubble formation is not well understood. The concept of bubble nuclei has gained acceptance in recent years. However, the definition of bubble nuclei is controversial and has ranged from "tiny" bubbles to "potential" for bubble formation (45). The advent of in-vivo bubble detection methods has confirmed that the appearance of circulating non-symptom-producing bubbles, or "silent bubbles" can indeed occur well before the clinical manifestations of DCS (35).

During the last decade, DCS research efforts have often used noninvasive precordial Doppler bubble detection techniques (30). DCS studies at the Armstrong Laboratory currently use a combination of echo imaging and Doppler for simultaneous visual and aural monitoring of precordial venous gas emboli (2). All subjects are monitored with the echo/Doppler to document the onset and degree of bubble formation (48).

Although the use of precordial echo/Doppler recording has great research value, its clinical use is very limited. It is difficult to correlate the occurrence of decompression sickness symptoms and the appearance of intravascular bubbles. Indeed, subjects frequently have large numbers of bubbles without complaining of symptoms and some have symptoms without any precordial bubbles. This is understandable because intravascular bubbles may not correlate with extravascular bubbles and it is believed that the extravascular bubbles produce most symptoms.

Right-to-Left Cardiac Cross-Over of Gas Emboli

Recent work at the Armstrong Laboratory has demonstrated that exposure to high altitude can result in the crossing-over of venous bubbles to the arterial side of the circulation (34). This condition can have serious clinical consequences. Although the cases recorded in these recent studies all occurred at altitudes below 30,000 ft, it is reasonable to assume that the cross-over phenomenon is more likely to occur as the altitude increases.

It is generally believed that most often inert gas bubbles evolve either extravascularly in the tissues, or intravascularly on the venous side of the circulation. Evolved venous gas emboli circulate through the right heart and are presumably filtered out in the pulmonary circulation (41). Although important in DCS research and in the progress of our understanding of the physiological mechanisms of this condition, VGE per se have not necessarily been considered clinically hazardous, unless in extreme numbers.

Arterial gas emboli (AGE), on the other hand, are generally viewed with great concern. Classic diver's air embolism can be fatal. Most often this condition is the result of pulmonary overexpansion during ascent in the water, forcing air into the pulmonary venous circulation, through the left heart and to the cerebral arterial bed resulting in blockage and ischemia (47). Another mechanism by which gas emboli could enter the arterial circulation and cause cerebral damage is by the crossing-over of gas emboli from the venous to the arterial side of the circulation. This may occur in the surgical setting, as well as the diving and aerospace situations (16, 37). Such "cross-over" to the systemic circulation is thought to occur by one, or a combination of the following routes (5, 36): 1) intracardiac septal defects, 2) large anatomical shunts within the lung parenchyma, and 3) the pulmonary microcirculation. Operationally, septal defects have been implicated in neurological DCS associated with diving (26, 52), but not with aviation (7).

A reversal of the normal left-to-right pressure gradient must occur in order for VGE to cross into the left heart. This right-to-left shunting can occasionally occur during quiet breathing. More profound reversals can occur upon release of a Valsalva, cessation of positive pressure breathing, the L-1 or M-1 anti-G straining maneuver, coughing, a Müller maneuver, negative pressure breathing during restricted inhalation, and during any situation that causes rapid and substantial venous return to the right heart (16). Most of these situations are common events in the operational aerospace environments.

In our recent study, left ventricular gas emboli were recorded with echo imaging in six volunteer subjects during exposure to simulated altitude. Five of the cases became symptomatic simultaneously with the time of AGE onset. The symptoms consisted of joint pain and skin mottling; no cerebral manifestations were reported. In all 6 cases, at the time of AGE onset, the VGE scores were high from all 5 monitored sites. Evaluation for intracardiac septal defects/ PFOs resulted in the following data:

(1) subject positive with PFO by Transesophageal (TE) Doppler
(1) subject positive with a small sinus venosis defect by TE
(1) subject negative for any defects by TE Doppler
(2) subjects negative by 2-D Echo Imaging only
(1) subject not available for evaluation

The common factor in all 6 cases was the high VGE load. The animal studies of Powell (35), Vik (46), and Butler (5) have demonstrated that cross-over will occur if enough VGE are generated. These studies demonstrated that as the venous gas load increases, overloading the vascular filtering mechanism, pulmonary arterial pressure (PAP) rises, arterial gas emboli start appearing, and neurological DCS and death can result. Since the rise in PAP is the triggering mechanism for cross-over, it follows that in order to get a large enough PAP rise to cause right-to-left slipover, there must be a large volume of VGE.

Since the results of this study showed that a septal defect was present in two subjects, but absent in another, at least two mechanisms may be involved in the cross-over phenomenon. Thus, whether by atrial septal defect (ASD), pulmonary microcirculation, or through A-V shunts, VGE can pass through the left heart, and be carried into the coronary, cerebral or systemic circulation. As a result, the use of ASD screening for air crew to reduce risk is not supported since the gas can cross-over through alternative channels. In addition, if TE is the best method for diagnosing ASDs, and since it is an invasive, expensive, and very uncomfortable procedure, such screening cannot be justified.

Cerebral arterial gas embolism is life-threatening and is thought to initiate a series of ill-understood complex physiological processes that are similar to those of a stroke (11, 12, 17). This new finding of a visualization of left-sided bubbles in human subjects at altitude points to an increased awareness of the potential seriousness of exposing people to altitudes that generate large numbers of VGE. A case can be made that altitude exposures that have been shown to repeatedly result in high venous gas loads in the majority of subjects should be avoided by aviators because the onset of cerebral symptoms in-flight is a subset of DCS that has the most serious consequences. Unlike the experimental situation in which subjects are monitored for intravascular gas, the pilot has no way of knowing if he/she has bubble formation until symptoms appear. Although pain-only DCS is not a life-threatening condition and is often ignored or simply not reported, cerebral symptoms such as blindness and altered states of consciousness can result in the loss of pilot and aircraft. The worst case scenario might be a situation in which a pilot is exposed for a period of time at 22,500 ft long enough to generate large numbers of VGE, at which point cabin pressurization is lost and the pilot is decompressed to 60,000 ft. It is likely that bubble growth will be massive and the PAP will rise rapidly resulting in cross-over of gas. This scenario is yet to be demonstrated.

Decompression Sickness above FL 400

Unlike the U-2, the new high-altitude fighter aircraft will presumably not have preflight denitrogenation procedures for protection against DCS. Yet it is likely that there will be a 5 psi differential pressurization schedule used, resulting in a cockpit altitude of about 22,500 feet. Thus, cabin altitudes may reach levels known to have significant DCS risk when exposure times exceed one hour (51). Furthermore, if an accidental rapid decompression occurs during such a flight, DCS risk may become paramount.

There are very few studies on DCS above 40,000 feet. In 1945, Sweeney (44) reported that "about 20% of the subjects decompressed to altitudes above 40,000 feet suffered bends during the ensuing five minutes at

altitude." Annis and Webb (1, 49) noted a DCS incident in one of their subjects within 5 minutes of being exposed to 80,000 feet simulated altitude, even though he had prebreathed for 3 hours. These subjects were wearing an elastic suit with positive pressure breathing capability. Results of a more recent study showed 50% DCS (n=4) in subjects at rest exposed to 40,000 ft for 2 hours (24).

DCS Latency vs. Altitude

It has generally been assumed that there is a short "grace" period (5 to 10 min) before DCS onset at any altitude above 30,000 feet due to the "inertia" of symptom onset. However, since essentially no hard DCS data for altitudes above 40,000 feet exist, the real onset times at these low pressures are unknown. For lower altitudes, if latency of DCS is plotted against altitude, there appears to be a linear relationship (32). Figure 2 shows the latency of DCS with no prebreathing. The data for this plot was obtained from the Armstrong Laboratory DCS Research Database (50), and a number of papers from the 1940s and 1950s (6, 9, 14, 18, 27, 43,). The results were screened to only include experiments that used mild exercise and defined the onset of DCS as the point at which mild symptoms were noted. There are no plots above 38,000 feet because usable data could not be found. When this band of latency is extended above 40,000 feet, it can be concluded that the time to onset of symptoms becomes "very short", probably shorter than was previously believed. Therefore, if the relationship in Figure 2 holds, there may be a significant DCS hazard in high-altitude "get-me-down" scenarios even with very short exposure times.
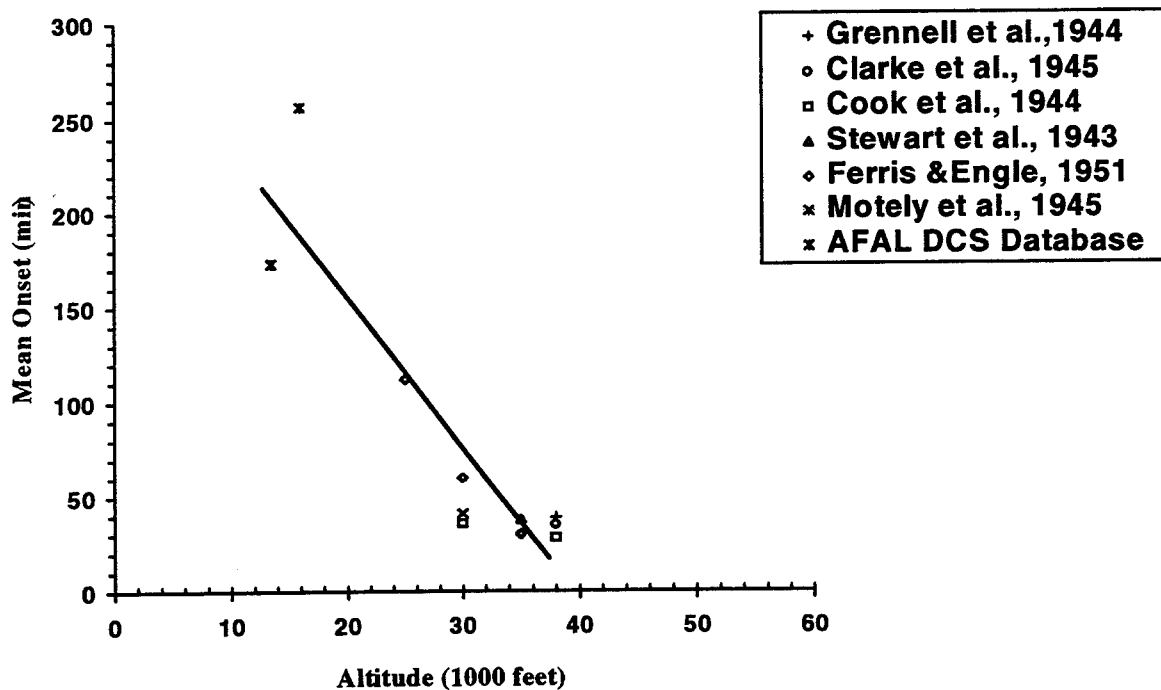
Figure 2. Altitude vs. time to DCS symptom onset.

Rapid Decompression and 'Get-Me-Down' Scenarios

The possibility of unplanned rapid decompression from loss of aircraft pressurization while at high altitude is of concern. In the flight scenario in which an unplanned rapid decompression to the ambient altitude of 60,000 ft is preceded by 1-2 hours of cruise at a cabin altitude of 22,500 ft, it can be assumed that bubbles were present prior to the rapid decompression. Thus, the moderate DCS risk associated with exposure to 22,500 ft will be magnified by the rapid decompression. In this scenario, existing gas emboli that slowly developed during the initial phase of the flight will rapidly expand during the rapid decompression phase, resulting in potentially serious symptoms with very short onset times--even shorter than those described in Figure 2. Even so, if recompression is rapidly initiated, and if the pilot stays conscious, chance for survival is excellent. This emergency situation is referred to as a "get-me-down" scenario. After loss of pressurization, the pilot is subjected to positive pressure breathing for hypoxia and descends to lower altitude as soon as possible, i.e., 1 to 2 minutes. Descending to below 10,000 ft will result in very low DCS risk, but under wartime conditions to perhaps very high threat from the ground (Figure 3). If the descent is to 25,000 ft, the DCS risk will be high because of the exposure at 22,500 ft and the RD. Of course, if the pilot breathes oxygen, he will have a lower risk than if the "normal" setting is used (Figure 4). If, because of ground threat, the pilot chooses to stay in the 35,000 to 40,000 ft altitudes, he will avoid the need for PPB, but his DCS risk will be very high and onset times short (Figure 5).
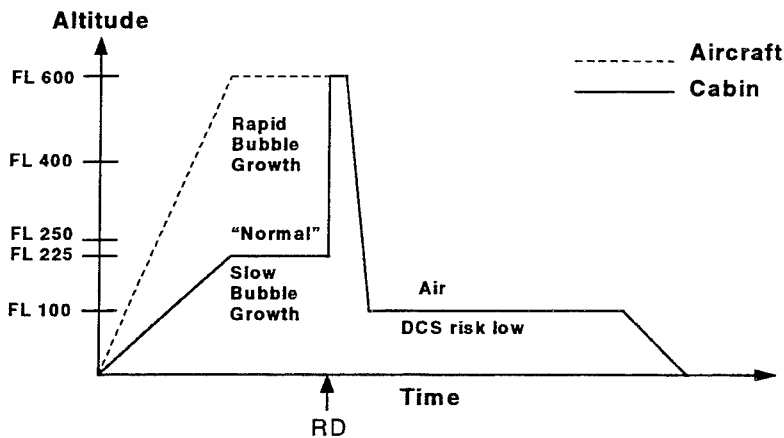
Figure 3. Get-me-down scenario with descent to 10,000 ft.
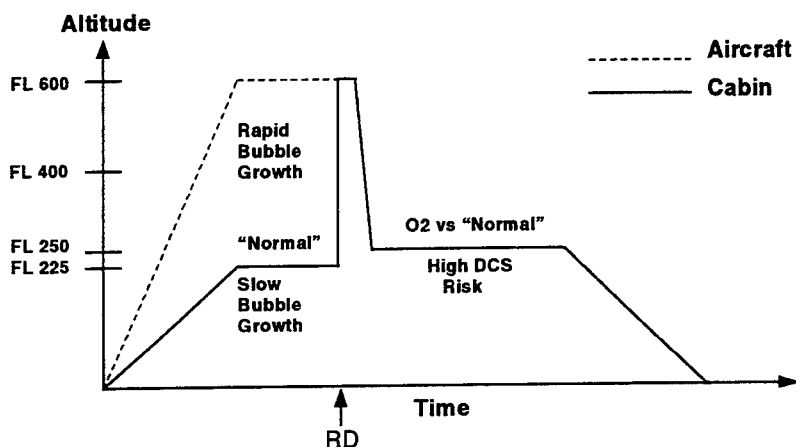
Figure 4. Get-me-down scenario with descent to 25,000
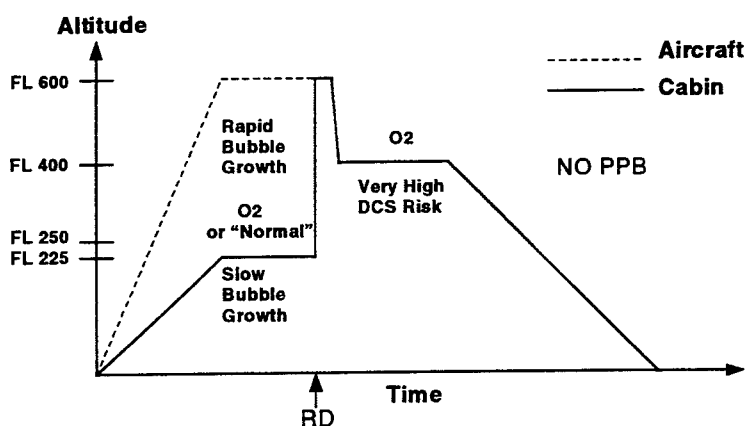
Figure 5. Get-me-down scenario with descent to 40,000 ft.

Sustained Exposure

Perhaps the highest DCS hazard is associated with potential situations in which descent to low altitude may not always be possible after an unplanned loss of pressurization (Figure 1) because operational constraints may overshadow physiological concerns, and may force the continuation of flight at the higher altitudes. Such sustained exposure could be accompanied by extremely high DCS risk, possibly some degree of ebullism (see below), and high levels of PPB to maintain consciousness (Figure 6). Therefore, determination of this DCS risk

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and latency at the higher altitudes is crucial to future fighter operations. If the risk is too great for sustained exposure at 60,000 ft, how long could the flight be sustained at 50,000 ft or some combination of descending altitudes? In a joint USAF/RAF Decompression Research Program, chamber protocols are currently being used to study the DCS and gas emboli risk for these scenarios.
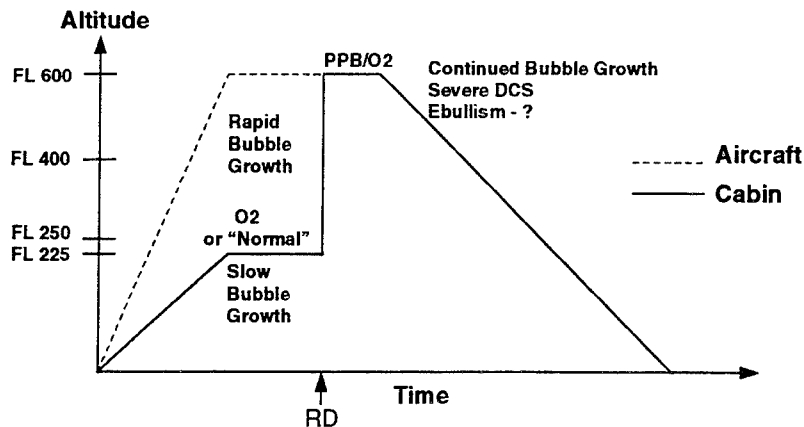
Figure 6. Sustained exposure at 60,000 ft.

Ebullism

If an aircraft was flying at altitudes higher than 63,000 feet and had an RD, an additional physiological hazard known as ebullism would occur in the unprotected or partially protected individual. Ebullism is the spontaneous boiling and degassing of body fluids and tissues as well as evaporative cooling and loss of body water, heat, and other materials (32). Conditions necessary for ebullism are present when the body is exposed to pressures below about 47 mmHg (63,000 feet or 0.91 psi) for a human with a core temperature of 37°C. This altitude is referred to as the Armstrong Line (53). However, due to variations of pressures and temperatures in the body, it is perhaps better to think of this limit as a band of altitude rather than a line. Ebullism in exposed human tissue may begin as low as 55,000 feet (39). When the vapor pressure of a tissue is reached, liquid changes to its gaseous state and gas cavities are formed in the tissue. Due to slight pressure and temperature differences among various tissues, the extent of these gas pockets will vary. Likewise, when recompression occurs, the gas pockets will spontaneously collapse into the fluid phase.

The pathophysiology of ebullism in animals was extensively studied in the 1950s and 1960s (3, 8, 13, 19, 21, 38). The animals in these studies were exposed to altitudes above 70,000 feet without protective equipment. Table 1 lists many of the effects of such exposures, some of which are prevented by the addition of positive pressure breathing.

Table 1. Some observed effects of ebullism in animals.

Severe tissue hypoxia immediately on decompression
Body volume doubles in 5-10 secs
Vomiting/defecation/urination in 5-10 secs
Unconsciousness/collapse in 9-12 seconds
Loss of voluntary control in 10 secs
Freezing of secretions (e.g. urine, salvia) by evaporation
Body temperature lowered by evaporation
Rapid increase in venous pressure
Circulatory arrest in seconds
Tonic and clonic seizures in 10-30 seconds
Apnea and spastic rigidity within 30 seconds
Total flaccid paralysis around 30 seconds

Nevertheless, survival is possible. Animals survive after exposure to hard vacuum from 90 to 210 seconds (3, 19, 21, 38). The significant factors influencing immediate survival are re-establishment of circulatory integrity and the degree of pulmonary and cerebral damage. The degree of damage to the lungs is critical to survival. Autopsies of animals exposed to vacuum universally show massive pulmonary damage that can range from petechiae to severe atelectasis to frank hemorrhage (13). Unless pulmonary exchange can be re-established, survival is not possible. If pulmonary exchange is possible, cerebral resuscitation may be successful depending on the exposure time.

Humans have been exposed accidentally to hard vacuum. There is one published case report in the literature of a prolonged unprotected exposure (22). In addition, several anecdotal reports of human exposures to vacuum exist. In these cases, people survived with limited or, in one case, no protection. During such unprotected exposures, the subjects lost consciousness rapidly and had varying degrees of injury, ranging from no significant symptoms to massive cerebral and pulmonary injury requiring intensive medical intervention. In the cases of partial protection (20), subjects described swelling and pain in exposed limbs. Whether these changes were severe enough to prevent a pilot from manipulating flight controls, or controlling the aircraft, is unknown. In one videotaped case (23), a subject was testing a space suit, lost suit pressure, and was instantaneously exposed to an altitude of 120,000 feet. He remembers the saliva boiling on his tongue prior to passing out, and then recalls the chamber monitor calling 14,000 feet as the chamber was being recompressed. He suffered no complications from this incident and was not hospitalized afterwards. The one published case history involved an individual who was exposed to approximately 74,000 for 3 to 5 minutes in an industrial accident (22). He was aggressively treated with hyperbaric oxygen and full ICU support. Neurological tests one year after the incident were above baseline levels. The authors did report the need to intubate the patient to avoid respiratory compromise due to frank pulmonary bleeding.

Since cerebral tissues tolerate low O2 tensions poorly, cerebral oxygen supplies must be re-established to avoid long term neurologic sequelae. In addition, cerebral tissue itself can undergo mechanical disruption by bubble formation. Despite these potential problems, most animal research reports show good recovery of neurologic function after as much as 2.5-3.5 minutes of exposure (21, 38) The industrial accident patient had decerebrate posturing and coma after his exposure; however, all neurologic measurements were returned to baseline levels within one year.

Future fighter aircraft may routinely fly at or above 63,000 feet. At present, crew protection against ebullism consists of cabin pressurization and full-coverage pressure suits. For example, in the U-2/TR-1 reconnaissance aircraft, a full-pressure suit is normally worn deflated. If there is an accidental loss of pressure, the suit automatically inflates. Full-coverage suits, however, severely limit mobility. In an effort to protect the pilot from both high-altitude exposure and high acceleration, yet retain mobility, integrated partial-pressure/G-suits

have been used. The disadvantage of these suits is that no protection is provided to the head or upper extremities, resulting in the potential for severe injury in the event of rapid decompression.

There is no medical treatment protocol for ebullism, probably because it is generally accepted that exposure to a vacuum is not survivable. That opinion is based, at least in part, on the results of the 1960s animal research. In addition, operational constraints dictated a fatalistic view. Survival was simply not considered possible. That view changed in 1982 when the industrial accident case history (22) was published describing the successful use of hyperbaric oxygen therapy.

The immediate objective is to re-establish pulmonary exchange. No research data are available. However, the use of PEEP and High Frequency Ventilation have been suggested for combating the massive atelectasis. Unless the lung is viable, death will result from cerebral anoxia. Other treatment objectives would appear to be best met by the use of hyperbaric oxygen therapy (HBO). The rationale for use of HBO for decompression accidents includes 1) bubble size reduction, 2) hyperbaric oxygenation of hypoxic tissues, 3) bubble resolution, and 4) reduction of neurological edema. However, recent work in our lab did not support this (42). It was found that ground level oxygen therapy was more effective than either HBO or ground level air breathing.

Conclusions

As a pilot's exposure altitude increases the incidence of DCS increases. Cabin altitudes in future high-altitude aircraft may reach levels known to have significant DCS risk. If an RD occurs, DCS risk will increase further. The altitude to which a pilot in a "get-me-down" scenario should descend to must be determined. If flight at these high altitudes must be continued after a rapid decompression, the DCS risk may become limiting. The new finding of left-sided bubbles in human subjects at altitude points to an increased awareness of the potential seriousness of exposing people to altitudes that generate large numbers of VGE. Exposure of unprotected or partially protected humans to altitudes above 63,000 feet results in tissue fluid vaporization, or ebullism. Survival can undoubtedly be improved with a better understanding of the pathophysiology of ebullism, improved protective measures, and the development of specific medical protocols.

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Discussion

DR. PILMANIS: In a recent study, we exposed animals to high altitude and the ebullism was severe. Unconsciousness occurred in less than 10 seconds.

DR. ACKLES: This is to what altitude?

DR. PILMANIS: We exposed about 200 animals to 87,000. It is severe in an unprotected animal. However, even minimal protection will reduce the effect. Certainly any amount of pressure breathing or mechanical compression of the body will improve the situation.

DR. GOODMAN: The potential bubble cross-over effects are very troubling. When you consider the rapid bubble growth that you projected at 60,000 feet and when you combine that with the enhanced cross-over effect, we might have to start thinking about the ramifications of high-level cardiovascular screening. What size bubble do you need to cross over into the coronary circulation? If you're getting some large bubbles into the coronary or cerebral circulation, we could be looking at a whole host of real serious problems?

DR. PILMANIS: I can't answer any of that, but the question goes on the list.

COL. STORK: I think a problem with the methodology is that you can't tell us about bubble volume.

DR. PILMANIS: True.

COL. STORK: We can only talk about the numbers that are present, but nothing about the size or the volume that's involved.

DR. GOODMAN: That's one of the problems that has to be sorted out. What is the critical bubble size before you have physiological problems?

DR. ACKLES: Have you done any measurements of pulmonary arterial pressure during the bubble growth?

DR. PILMANIS: We're about ready to start doing that. The technology is improving, but the methods are difficult. I just received a Russian paper where they did invasive measurements at altitude. When venous gas emboli occurred during altitude exposure, pulmonary arterial pressure did go up significantly.

DR. ACKLES: To the cross-over effect?

DR. PILMANIS: They did not mention cross-over to the arterial side of circulation. In another study, the bubbling reached a point where all the sheep died from air embolism. So if you carry it far enough, it does become quite dangerous. The question is whether we are in that range or are we well below that range?

SQN LDR RYLES: What we're hoping to do is to use 2D echocardiography to non-invasively measure pulmonary arterial pressure and at the moment, we're in the phase of assessing how effective that measure is. It might not have the resolution that we need. The only method that's been successfully used is indwelling lines, which is not what we would prefer.

PROF. ERNSTING: I would like to introduce a general point. From the intravascular bubble formation point of view, it seems a little unrealistic to talk about 60,000 feet exposures, because most of our breathing systems maintain the absolute pressure in the lungs of 120-130 millimeters of mercury and that's going to be transmitted directly to the heart. I don't see the pressure inside the heart chambers dropping below that absolute pressure because in most pressure suit systems, you have trouble getting rid of gas in a rapid decompression. I suppose one place that the pressures would drop close to ambient would be on the venous side of circulation. It's interesting to think what happens to those bubbles as the blood flows into the venous reservoirs and breathing pressure increases

when you're exposed to 60,000 feet, do the bubbles get smaller again because the pressure has gone up? The absolute pressure in that blood is going to have to come back to the baseline level. I feel as you do, that it's bubbles outside the central circulation that causes the problem.

DR. PILMANIS: Except for bubbles that cross over to the arterial side. I worry about that, but that does require a fairly severe condition before occurring. Under those circumstances the chances are good that they will lose consciousness rapidly.

COL. SHERMAN: Would it be possible to develop a computer-monitored life-support system integrated with a automatic recovery system like the G loss of consciousness (GLOC) system? For example, if you were flying an F-22 and you had experienced a decompression where imminent incapacitation would be expected because the pressure assembly was inadequate to maintain consciousness, would it be possible to develop an automatic recovery system which would bring the aircraft to a lower altitude on a preselected course until the pilot recovered. I realize this is a question that only applies to future aircraft and depends on whether the policy makers will fund the concept.

LT. COL. DEMITRY: The automatic recovery system for G loss of consciousness is not robust, but it certainly has been demonstrated many times and is credited with saving multiple lives in flight tests.

DR. PILMANIS: I suppose the question is whether the need to be exposed to those flight conditions is sufficiently critical to risk loss of consciousness which would lead to the development of a recovery system or should you avoid the situation in the first place? Another trade off.

LT. COL. DEMITRY: I have a few comments that are not fully independent of your question? We really do need a computerized model, that takes in all the different operational conditions and provides the protection automatically. I view it as an operator; I want automatic fuel flow so that I don't have to worry about fuel management. I don't want to have to worry about my life-support systems. If I'm going high, and we're going there, the life support systems must keep up with the aircraft technology. Before we can automate anything, however, we need a model based upon empirical data. We know what the flight control systems can do. We know about fuel flow and propulsion. We need to know what we can expect from the life-support system before going into that environment.

COL. STORK: Let me take just a moment and summarize what I think we've covered this morning. There were several salient points that immediately come to mind: we're presently flying above 50,000 feet and we're doing that with inadequate protection of the aircrews; the F-22 and future aircraft will routinely fly above 60,000. Except for the F-22, there are currently no requirements for a life-support program in the Air Force focused towards high-altitude protection; one of the major accomplishments is that we've finally reached some agreement on oxygen delivery equipment and standards, thanks in good part to the efforts of Dr. Ernsting. Although there is good agreement on minimal standards for FIO_2, there's still some discussion on what the maximum FIO_2 should be as regards the operational importance of atelectasis; the DCS risk will be significantly increased at altitudes above 50,000 feet and there is the additional complication of ebullism above flight level 630 and how that might lead us to more seriously consider the need for full- or partial-pressure suit protection. Whether the problem is ebullism or decompression sickness or oxygen delivery schedules or PPB or whatever, it's the integrated aggregate of problems we need to be concerned with. We need to be focusing on the human system with all of the complications of the life-support equipment, and how that performs within the weapons system ,and how that weapons system is employed. It is very clear that we can't afford to look at any one particular problem in isolation.

High Altitude Breathing Systems and Requirements

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The primary purpose of oxygen equipment is, of course, to prevent the hypoxia associated with ascent to altitude, to whatever level. In achieving this aim, however, a number of factors must be considered, both of a physiological nature and in general terms. This paper therefore describes the theoretical physiological and general requirements of oxygen systems, and some aspects of current equipment design and compliance.

Physiological Requirements of Oxygen Systems

Provision of adequate oxygen concentration at adequate pressure. In order to prevent hypoxia, alveolar oxygen tension (P_AO_2) must, if possible, be maintained at its sea level value of about 103 mm Hg. This can be achieved by a progressive enrichment of inspired air with oxygen, at the expense of nitrogen as altitude increases (termed Airmix). Eventually, the inspirate will be composed entirely of oxygen and this point is reached at an altitude of 33,700 feet, where atmospheric pressure (P_B) is 190 mm Hg: $P_AO_2 = 103$ mm Hg, $P_AH_2O = 47$ mm Hg, and $P_ACO_2 = 40$ mm Hg.

Thus, P_AO_2 at 33,700 feet when breathing 100% oxygen will be the same as P_AO_2 at sea level when breathing air: an example of the concept of Equivalent Altitudes.

At altitudes above 33,700 feet, even 100% oxygen will not prevent a fall in P_AO_2 but, because of the significance of the shape of the oxygen dissociation curve, severe hypoxia will not begin to develop in healthy individuals until P_AO_2 has fallen to below about 54 mm Hg. This may be predicted to occur at an altitude of about 40,000 feet where: $P_B = 141$ mm Hg: $P_AH_2O = 47$ mm Hg, $P_ACO_2 = 40$ mm Hg and $P_AO_2 = 54$ mm Hg. An altitude of 40,000 feet (equivalent to an altitude of 10,000 feet when breathing air) is the maximum to which ascent may safely be made, even when breathing 100% oxygen, and 141 mm Hg should therefore be regarded as the "ideal" minimum to which total lung pressure can safely be allowed to fall.

At altitudes above 40,000 feet, 100% oxygen under positive pressure (Pressure Breathing for Altitude protection (PBA)) must be delivered in order to prevent hypoxia; and, in order to maintain total pressure within the lungs at the minimum acceptable level (i.e., at 141 mm Hg: the 40,000 feet equivalent), the magnitude of positive pressure breathing delivered must increase progressively with altitude. So, for example, at an altitude of 45,000 feet (where $P_B = 111$ mm Hg: $P_AO_2 = 39$ mm Hg, $P_AH_2O = 47$ mm Hg, and $P_ACO_2 = 25$ mm Hg) a positive pressure of 30 mm Hg will be required. Clearly, even higher levels of pressure breathing are required as altitude increases.

Pressure breathing is not without considerable physiological penalties of its own, however, both of a direct nature and on the cardio-respiratory system. Consequently, at very high altitudes, the level of pressure required to prevent hypoxia completely must be balanced against the potential disadvantages of that pressure. In other words, a compromise must be reached between the magnitude of pressure breathing that is physiologically tolerable and an acceptable degree of hypoxia. Furthermore, it is important to appreciate that PBA is an emergency procedure only, and that the physiological acceptability of such protection is predicated on the assumption that descent to a safer altitude will be (and must be) initiated immediately the emergency occurs.

It is as an attempt to minimize the potentially harmful physiological effects of positive pressure breathing that counterpressure garments are employed to support the chest, abdomen and limbs. These so-called partial-pressure assemblies become more extensive the higher the altitude to which the aircrew may be exposed, such as when even higher levels of pressure breathing are required, or indeed if the breathing gas is other than 100%

oxygen (such as the 94% provided by first generation molecular sieve oxygen concentrators). For example, an oronasal mask, although capable of holding a positive pressure of 100 mm Hg, is suitable for use by itself with pressures of up to only 30 mm Hg. When used in conjunction with a sleeveless but conventional upper pressure garment (such as the venerable RAF partial-pressure jerkin (PPJ)), a maximum pressure of 60 mm Hg can be tolerated, while the combination of mask, PPJ and lower pressure garment (*ie* G-trousers) will allow 70 mm Hg to be tolerated at an altitude of 60,000 feet. At pressures above this (and up to 100 mm Hg), head and neck discomfort dictate the need for a partial-pressure helmet (together with upper and lower pressure garments) to apply breathing pressure not only to the respiratory tract but also to the ears, the neck and the floor of the mouth. If exposure to altitudes that would require the protection of more than ~100 mm Hg positive pressure is possible, then a full-pressure suit must be worn. The implications for these requirements of the routine use of pressure breathing for G protection (PBG) and the consequent need to reduce the weight and bulk of protective garments, are discussed elsewhere.

Figure 1 summarizes the relationship between the altitude to which decompression occurs, and the time imposed to achieve a safe descent for these combinations of partial-pressure equipment. All three combinations of equipment and pressure breathing levels will provide protection against the effects of loss of cabin pressurization up to the indicated cabin altitude, provided that immediate descent at the maximum rate (10,000 feet per minute) is undertaken to below 40,000 feet.
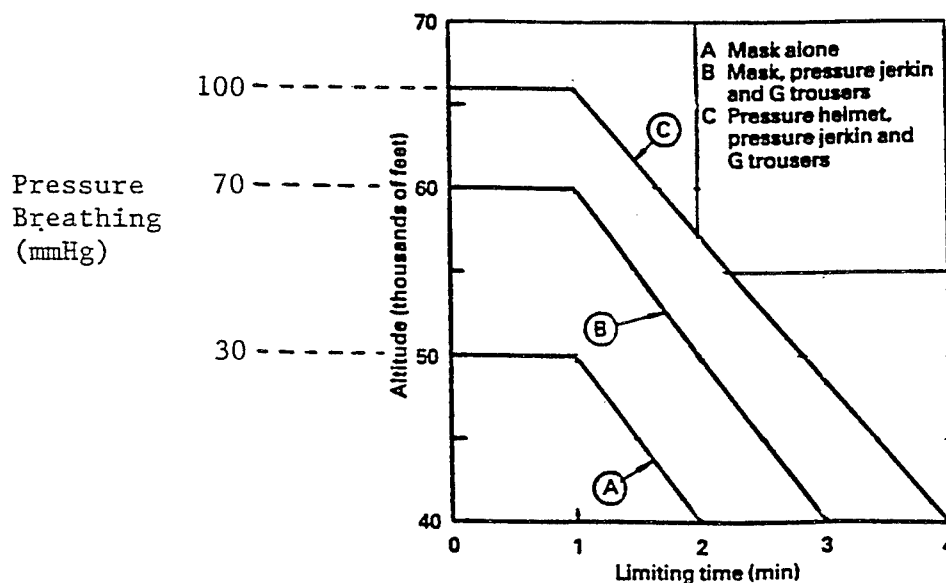
Figure 1. Relationship between DCS onset altitude and time required for safe descent under 3 equipment conditions.

The physiological requirements for oxygen on ascent to altitude may be summarized thus:

0 - 8,000 feet	Air
8,000 - 33,700 feet	Air enriched with oxygen
33,700 - 40,000 feet	100% oxygen
above 40,000 feet	100% oxygen under pressure

In practice, pressure breathing usually commences when cabin altitude exceeds about 38,000 feet. Furthermore, inspired oxygen concentration prior to a rapid decompression must be sufficient to prevent hypoxia occurring immediately after the event.

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Provision of Adequate Nitrogen. In order to avoid acceleration atelectasis, the inspired gas should contain at least 40% nitrogen, provided that the requirements to protect against hypoxia are not compromised.

The physiological requirements for inspired gas composition described allow the construction of a cabin altitude *vs* oxygen concentration diagram of the sort shown in Figure 2.
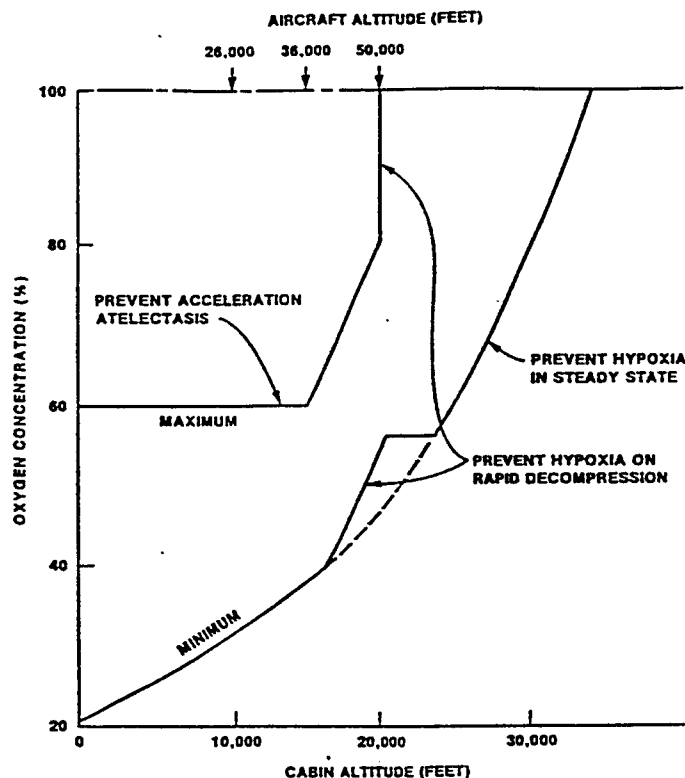
Figure 2. Cabin altitude vs. oxygen concentration with regard to prevention of atelectasis and hypoxia.

In this figure, the lower curve represents the minimum concentration of oxygen required to prevent hypoxia, while the upper curve represents the maximum oxygen concentration acceptable if acceleration atelectasis is to be avoided. The kink in the "hypoxia curve," the precise position of which will vary with the cabin pressurization profile of the aircraft (i.e., with the relationship of cabin altitude to aircraft altitude (top axis), reflects the additional oxygen concentration in the inspired gas required to prevent hypoxia should a rapid decompression take place from within that band of cabin altitudes. Similarly, the increase in the upper curve from 60% oxygen at a cabin altitude of about 15,000 feet to 80% (in this example) and then to 100% by 20,000 feet reflects the need to breath 100% oxygen on, or immediately after a rapid decompression. Such a diagram can be used by engineers responsible for the construction of the breathing system as the basis for design, since the described limits indicate the band between which oxygen delivery to the user must be controlled.

Provision of Adequate Ventilation and Flow. Current (ASCC) requirements state that military oxygen systems for use by aircrew should accommodate peak instantaneous flows of 200 L(ATPD).min^{-1}, at a maximum rate of change of 20 L(ATPD).sec^{-2}. Furthermore, the system should be able to deliver a respiratory minute volume of at least 60 L(ATPD). There is evidence to suggest that while peak inspiratory flows in flight may occasionally exceed 200 L(ATPD).min^{-1} during air combat maneuvering, minute volumes as high as 60 L(ATPD) are seldom achieved. The greatest demand placed on a breathing system is on the ground prior to take-off: unfortunately, breathing system performance is usually at its worst in this situation.

Imposition of Minimal Added External Resistance. Added external resistance, whether it be in the inspiratory or expiratory phase of the respiratory cycle will produce unwanted physiological effects, including a

reduction in minute volume, an increase in the work of breathing, hyperventilation, and feelings of asphyxia. It is therefore most important that the external resistance imposed by a breathing system be kept as low as possible, and so allow respiratory demands to be met. Thus, the opportunity must be taken whenever possible to reduce sources of added external resistance. Low pressure flow pathways (known to be the main culprits responsible for added resistance) should ideally be wide-bore and smooth-bore with as few acute bends as practicable, and the design of oronasal mask valves should be optimized within the constraints of providing the other required features of military breathing systems.

Dispersal of Expirate. The breathing system must be designed so as to disperse expired carbon dioxide to ambient, to keep added dead space as low as possible (^{2} 600 ml) in order to avoid significant rebreathing, and to allow the delivery of 100% oxygen as soon as possible after rapid decompression.

General Requirements of Oxygen Systems

Safety Pressure. A slight but continuous overpressure (safety pressure) in the system will ensure that any leaks from it (most commonly from an ill-fitting mask) will be outboard, ensuring that alveolar oxygen tension is uncompromised, particularly when above 10,000 feet.

Protection Against Toxic Fumes and Decompression Sickness. The ability to select 100% oxygen manually at any altitude provides protection should cabin air become contaminated by toxic fumes, and should decompression sickness be present or a possibility.

Evaluation of Integrity, Indication of Supply and Flow, and Indication of Failure. It should be possible for the user to establish the integrity of the system before take-off by means of a simple test (for example, by manually selecting the pressure breathing facility for a brief period). Once in use, a visual indication of gas supply and flow should be available to the user. Furthermore, any degradation of the system in flight should be made apparent to the user immediately, either by some subjective means (such as an added resistance to breathing if a disconnect occurs) or by an objective indication (such as a low pressure warning light).

Convenience. As far as possible the system should be simple to use, and preferably automatic.

Duplication. In those aircraft in which the oxygen equipment provides the primary protection against hypoxia (i.e., in those with low differential pressure cabins), a degree of redundancy is essential. In modern systems this is frequently achieved by providing a standby or secondary breathing regulator for use should the primary device fail. An alternative and independent oxygen supply (Emergency Oxygen or "EO") should also be provided for use if the primary supply fails or is contaminated.

Protection During High-Altitude Escape. The possibility of an escape from a military aircraft at high altitude requires the provision of an oxygen supply for use after abandonment during descent to below 10,000 feet. Such a supply, which usually doubles as the EO, must obviously leave the aircraft with its user, and so is commonly mounted on the ejection seat.

Independence from the Environment. All items of oxygen equipment must function satisfactorily in the various extreme environmental conditions met with in flight: that is, conditions of pressure, temperature (especially cold), acceleration, vibration, and windblast. In this respect, cognizance of the requirement for NBC protection is also necessary.

Current Systems

A brief consideration of the evolution of oxygen systems over the past half century will give an indication of the extent to which these "ideal" requirements have been realized--and to what extent improvement is still needed.

Until the early 1940s, oxygen delivery to aircrew was by means of a simple continuous flow system from a gaseous oxygen storage cylinder to a valveless oronasal mask. Such systems were then widely replaced by the more efficient and effective Economizer system, in which a non-rebreathing reservoir was interposed between the source of breathing gas and the user. The late 1940s saw the introduction of pressure demand oxygen systems, with the breathing gas (as 100% oxygen or as "airmix," by injection air dilution) being delivered only on demand via mechanical panel-mounted regulators. These devices also provided safety pressure automatically above 10,000 - 12,000 feet, and pressure breathing, again automatically, above 38,000 feet. Variants were later built to accommodate the increased pressure breathing delivery characteristics required for use with partial-pressure clothing at altitudes above 45,000 feet.

A further refinement, in the UK, was the introduction in the mid 1950s of the P/Q series of oxygen masks with their increased comfort, stability under +Gz acceleration, and excellent sealing properties during pressure breathing. These masks incorporated a non-return inspiratory valve and a pressure-compensated expiratory valve. The overall resistance to breathing from these systems, however, remained quite high; although the later introduction in British aircraft of personal equipment connectors (PECs)-- the means by which pneumatic and electric supplies to the pilot were delivered via a single-point mechanism-- facilitated greater use of lower resistance wide and smooth bore anti-kink hosing. The use of a PEC also allowed the EO supply to be plumbed in permanently, so dispensing with the need to connect it, and its complex pull-off inward relief/excess pressure relief device, directly to the mask hose during the strapping-in procedure. The EO system could also be more sophisticated, even to the point of mimicking the pressure demand characteristics of the primary system.

The oxygen systems installed in USAF fast jet aircraft have remained relatively unchanged since the 1950s, with most still incorporating pressure demand panel-mounted regulators. The design has been refined considerably over the years so that the current CRU series of "narrow" regulators occupy less panel space and have improved performance characteristics. A seat-mounted continuous flow EO system is still used, with delivery via a pull-off inward relief/excess pressure relief connector. The early A13A oxygen mask was succeeded by a series of MBU masks with improved sealing capabilities. The combined inspiratory and compensated expiratory valve fitted to some of these masks did, however, impose considerable resistance to breathing; as does the delivery hose arrangement in even the most modern assemblies (*e.g.*, TLSS and COMBAT EDGE).

With increasing avionic sophistication came the need to make available as much console space as possible: for life-support engineers, this meant relocating elements of the oxygen system. The use of pneumatic engineering allowed miniaturization of pressure demand regulators to the extent that they could be practicably mounted on the man. In the UK, the first such device was introduced in the late 1960s to replace the American 100% oxygen regulator system supplied with the F4 aircraft purchased for the RAF and RN. Miniature man-mounted regulators successfully equipped a generation of British fast jet aircraft, but were not without problems.

For all subsequent UK aircraft, therefore, so-called duplex seat-mounted pressure demand oxygen regulators have been procured, integrated with a PEC, and comprising a main and a standby regulator. Control is based on pneumatic principles with a pilot valve governing flow of oxygen through a main demand valve, while gas loading of the breathing diaphragm provides safety pressure and pressure breathing. The main regulator provides the usual airmix facility to 33,000 feet with safety pressure from 15,000 feet; while the standby regulator delivers 100% oxygen under safety pressure at all altitudes. This duplication of regulators provides a high degree of redundancy, and so enables a mission to proceed even when the main breathing device has failed. A gas-loaded air inlet will impede inspiratory effort in the absence or failure of the breathing supply, so providing the all-important and immediate subjective indication of something amiss. A seat-mounted oxygen regulator also allows relatively simple incorporation of an EO supply.

Despite the considerable disadvantages of using liquid oxygen (LOX) as a source of breathing gas, LOX has been the storage method of choice for many decades by virtue of the savings it offers in terms of weight and bulk. But now, molecular sieve technology has reached maturity, and its potential for use in military aircraft is being realized. This important topic is discussed elsewhere, but it must be emphasized that a Back-Up, Auxiliary, or Stand-By supply of gaseous oxygen (BUO, AOS, SBO) is still highly desirable in current Molecular Sieve Oxygen Concentrator (MSOC) systems.

Thus, there has been a steadily progressive evolution of oxygen systems for high-performance aircraft over the past few decades and, while national differences do exist, there are some clear generalizations best shown by summarizing the leading generic particulars of systems fitted to some current aircraft.

Example 1   F15, F16 [conventional]

- LOX with no stabilization
- single, panel-mounted pressure demand regulator
- delivery of airmix and 100% oxygen
- delivery of safety pressure and pressure breathing
- continuous flow, seat-mounted EO supply delivered via quick-release pull-off mask hose connector
- relatively high resistance to breathing because of hose, connectors and combined valves in mask

Example 2   Tornado, Hawk

- LOX with stabilization
- duplex, seat-mounted pressure demand regulator, integrated with a PEC
- delivery of airmix and 100% oxygen via lockable mask-hose connectors
- delivery of safety pressure and pressure breathing
- seat-mounted EO supply delivered via standby regulator
- relatively low resistance to breathing because of wide PEC ports, wide, smooth bore anti-kink hose, and low resistance mask valves

Example 3   Harrier GR5

- MSOC
- duplex, seat-mounted pressure demand regulator, integrated with a PEC
- delivery of airmix and 100% oxygen via lockable mask-hose connectors
- delivery of safety pressure and pressure breathing (incl PBG)
- seat-mounted EO/BUO supply delivered via standby regulator
- relatively low resistance to breathing because of wide PEC ports, wide, smooth bore anti-kink hose, and low resistance mask valves

Example 4   Eurofighter [final system]

- MSOC
- seat-mounted Aircrew Services Package, comprising duplex pressure demand regulator and anti-g valve, integrated with a PEC
- delivery of airmix and 100% oxygen via lockable mask-hose connectors
- delivery of safety pressure and pressure breathing (incl PBG)
- seat-mounted EO/BUO supply delivered via standby regulator
- relatively low resistance to breathing because of wide PEC ports, wide, smooth bore anti-kink hose, and low resistance mask valves

Example 5   F22

- MSOC
- panel-mounted pressure demand regulator with integral anti-g valve (BRAG)
- delivery of airmix and 100% oxygen
- delivery of safety pressure and pressure breathing (incl PBG)
- seat-mounted EO supply delivered via dedicated mini demand regulator
- No BUO supply
- moderate resistance to breathing because of hose and connectors

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The facilities provided by current oxygen systems represent the distillation of much experience with many configurations of various components. The duplication of the pressure demand regulator, in particular, and the associated arrangement of controls, has provided greater redundancy, improved physiologic performance (especially with respect to breathing resistance), and enhanced operational capability. But this has been achieved at the expense of simplicity of design and construction, and at considerably higher financial cost.

Discussion

COL. SHERMAN: Maj. Neubeck, do you have a chart showing the pressurization curve for the F-22, or what the schedule is going to be?

MAJ. NEUBECK: No, sir, I don't.

MS. MCGARVEY: We can present one. It is just like the schedule in the F-15 and F-16 aircraft. It's an isobaric schedule that starts at 8,000 feet and maintains a 5 PSI differential to altitude.

Evolution and Operational Use of
Molecular Sieve Oxygen Concentrator Systems

George W. Miller, Major, USAF (Ret)
1Lt Jerold E. Fenner

Introduction

The purpose of this paper is to review the development of molecular sieve oxygen concentrator systems. Emphasis will be placed on US Air Force systems. Oxygen concentrator performance, operational use, and backup oxygen system capacity will be discussed. Further, potential future technologies which may be applied in aircraft oxygen systems will be mentioned.

Presently, molecular sieve oxygen concentrator (MSOC) technology is routinely applied on military aircraft for the generation of an oxygen enriched breathing gas to prevent hypoxia. MSOCs use a technique known as pressure swing adsorption (PSA) to separate oxygen from engine bleed air. This technique has grown in popularity in recent years because of its simplicity, reduced energy consumption, and low operating costs when compared to conventional liquid oxygen systems. Use of MSOC technology on military aircraft eliminates the liquid oxygen logistical requirements and safety issues, decreases aircraft turn-around time, allows more options for aircraft basing, extends mission time if limited by the quantity of liquid oxygen, and significantly lowers operational costs. Although many types of oxygen generating systems, such as permeable membrane, electrochemical, organic chelate, and ceramic have been investigated for aircraft use, only MSOC technology has been installed on production aircraft.

MSOC systems and liquid oxygen systems differ in several fundamental characteristics. Liquid oxygen systems contain a finite amount of oxygen which can limit mission duration. MSOC systems will continue to supply enriched oxygen if sufficient engine bleed air pressure is delivered to the concentrator. This pressure can vary significantly during the different phases of the mission. Liquid oxygen systems are self-contained and independent from other aircraft systems. MSOC systems must be integrated into the aircraft environmental control system. Generally, the goal of this integration is to minimize MSOC weight and space and maximize performance. Liquid oxygen systems can deliver 99.5-100% oxygen on demand simply by switching the aircrew regulator to the 100% mode. Current operational molecular sieve oxygen concentrators are limited to a maximum oxygen concentration of up to 93-95%. Further, this purity can only be delivered under certain operating conditions.

Typically, MSOCs are comprised of two or three beds or canisters of molecular sieve adsorbent, valving, a purge orifice, and an electronic timer (Figure 1). In the PSA process, the adsorbent beds are alternately cycled through steps of adsorption and desorption. During the adsorption step, air at moderate pressure (20-60 psig) enters the adsorbent bed, whereupon nitrogen is preferentially adsorbed and enriched oxygen is recovered from the MSOC product port. During the pressurization step nitrogen is adsorbed because the molecular sieve has a greater affinity for the nitrogen molecule. The adsorption step is followed by desorption, or venting, of the adsorbed nitrogen to ambient pressure. The ambient pressure varies with the aircraft altitude. This swing in pressure reverses the adsorption process. At ambient pressure the molecular sieve can retain only a small portion of the nitrogen, hence, most nitrogen is released. During the desorption step, a small portion of the product oxygen flows through the orifice to the depressurized bed to purge the remaining nitrogen from the bed. This phase of the process prepares the molecular sieve for the next pressurization step. These cycles of adsorption and desorption are continuously repeated resulting in a stream of enriched oxygen at the outlet of the oxygen concentrator.

MSOC system performance is affected by intrinsic and extrinsic factors. Intrinsic factors are determined by the specific MSOC design. Several intrinsic factors are the type and quantity of molecular sieve, concentrator

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cycle time, and purge flow. The quantity of molecular sieve determines the amount of nitrogen which can be adsorbed during the pressurization step. Hence, a concentrator designed for greater oxygen flow (or more crew members) will require a greater quantity of molecular sieve. Also, a faster (or shorter) cycle time will improve MSOC performance while a slower (or longer) cycle time will lower performance. At a slower cycle time the nitrogen wavefront will penetrate deeper into the molecular sieve and a higher nitrogen concentration will be measured in the product gas. Purge flow is determined by the orifice diameter and must be optimized for the specific concentrator. Extrinsic factors are determined by the specific characteristics of the aircraft engines, environmental control system, and mission profile. Several extrinsic factors are inlet air pressure and temperature, ambient pressure or altitude, and product flow rate or the demand placed on the system by the aircrew. MSOC performance generally improves with increasing inlet air pressure. Also, operation near room temperature improves performance, where as, temperatures significantly above or below room temperature generally lower system performance. Further, system operation at higher altitudes (or lower ambient pressures) generally results in improved system performance. An additional characteristic of molecular sieve oxygen concentrators is that at low outlet flows (~5-15 LPM) the oxygen concentration remains nearly constant at about 93-95%, however, at higher flows the oxygen concentration waveform is nearly sinusoidal. The amplitude of the waveform increases as the product flow increases. When MSOC performance is depicted graphically, the average oxygen concentration of the sinusoidal waveform is given. The average oxygen concentration is generally determined by averaging the maximum and minimum of the waveform.
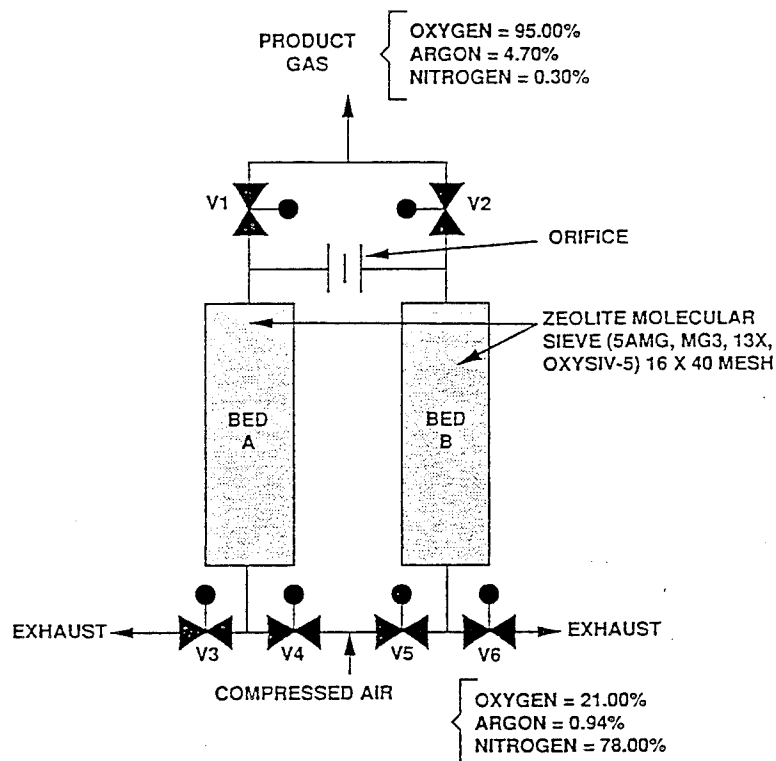
Figure 1. A Standard Molecular Sieve Oxygen Concentrator.

Presently, most MSOCs use either 5AMG or OXYSIV-5 molecular sieve adsorbents. 5AMG contains 5A molecular sieve crystallites while OXYSIV-5 and MG3 contain 13X crystallites (Figure 2). These molecular sieves are manufactured by UOP, Des Plaines, Illinois. Both molecular sieves are synthetic crystalline zeolites possessing a uniform crystal framework and, hence, apertures or pores with precise dimensions. The basic building blocks of the crystal are SiO_4 and AlO_4 tetrahedra with exchangeable cations. The type of framework and exchangeable cation will determine the dimension of the crystal pores. The presence of these ions gives rise to the strong electrostatic fields within the crystal lattice.
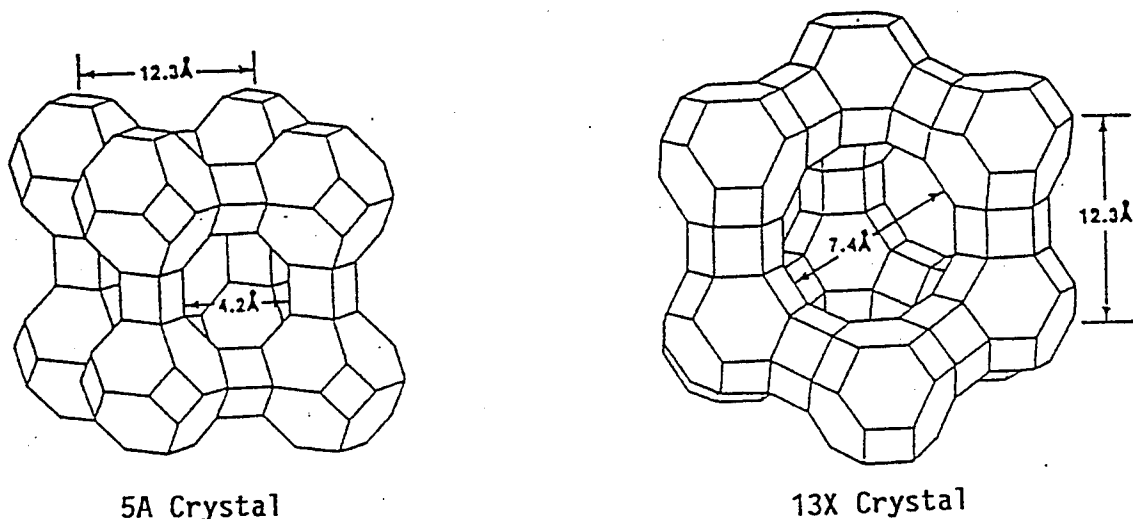
Figure 2. Structure of 5A and 13X Molecular Sieves.

The degree to which a molecule adsorbs within a particular molecular sieve is primarily determined by the temperature, pressure, and the adsorbate molecule's kinetic diameter, polarity, and degree of unsaturation. PSA separation of nitrogen and oxygen by either 5A or 13X based molecular sieves is possible due to a difference in molecular polarity. Nitrogen is adsorbed in greater quantity because of the favorable interaction between the quadruple moment of the nitrogen molecule and the electrostatic fields of the crystal framework (Figure 3). Oxygen and argon molecules are nonpolar and adsorb in nearly identical quantities. Hence, in current MSOC systems both oxygen and argon are concentrated resulting in the oxygen purity limitation of 93-95% (the remainder is mostly argon). In the PSA process the molecular sieve is not chemically altered because only physical adsorption occurs. Further, the heat of adsorption produced when nitrogen adsorbs within the molecular sieve during the high pressure step results in a slight temperature change (4-5°C) during operation.

Although both molecular sieve crystallites 5A and 13X are alkali metal aluminosilicates, they differ in crystal structure, pore size, and exchangeable cation. The 5A molecular sieve has a type A framework with calcium cations and a pore free aperture of 4.2 Angstroms. The 13X molecular sieve has a type X framework with sodium cations and a pore free aperture of 7.4 Angstroms. In Figure 2 oxygen ions are located at the midpoint of each line and either a silicon or aluminum ion is positioned at the intersection of the lines. The exchangeable cations are not shown but would be distributed inside the central cavities. Molecules with a kinetic diameter less than the pore free aperture will readily traverse the pore channels and adsorb to some degree within the volume of the large central cavities. Those molecules with a kinetic diameter greater than the pore free aperture will be unable to enter the crystal structure. Based on this characteristic, molecular sieves with the larger pore aperture (13X crystallites) have shown a greater ability to extract chemical contaminant molecules from the engine bleed air.[1] Hence, contaminants are prevented from exiting the MSOC with the product oxygen.

To prevent destruction of the crystal framework in an operating MSOC due to severe pressure fluctuations, the crystals (average size of 1 to 4 μm on edge) are combined with an inert clay binder to form pellets. The pellet size for most molecular sieves used in MSOCs is 16 X 40 mesh. Further, the pellets must be retained properly within the canister to prevent their movement and possible attrition. Generally, this retention is accomplished with an arrangement of metal screens and coil springs. Improper bed retention (either too low or too high a retention force) will result in molecular sieve breakdown or "dusting." Hence, the proper design of the retention system is critical to the MSOC design. In addition, most MSOC systems have a particulate filter at the outlet of the oxygen concentrator to prevent particulates from entering the aircrew regulator should dusting occur.

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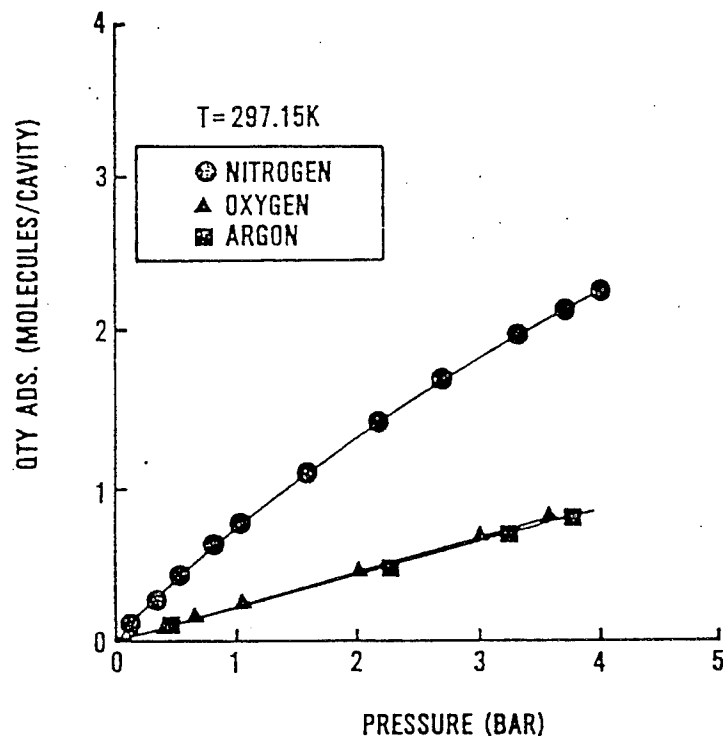
Figure 3. Adsorption Isotherms for Nitrogen, Oxygen, and Argon on Molecular Sieve.

Historical Background

Zeolites were first recognized by Cronstedt as a separate group of minerals in 1756.[2] These minerals appeared to boil when heated. Actually upon being heated these hydrated minerals release trapped or adsorbed water. Cronstedt called the minerals zeolites which in Greek means "a boiling stone." The term "molecular sieve" was originated by McBain in 1932.[3] McBain noticed that certain minerals, primarily dehydrated crystalline zeolites, acted as sieves at the molecular level. These materials had a great deal of internal volume available for adsorption which was only accessible by a network of channels or apertures. Molecules small enough to enter the channels can access the internal volume where the crystal adsorption sites are located.

The first definitive experiments to show that zeolite minerals could be used to separate gas mixtures were conducted by Barrer in 1945.[4] He classified zeolites on their ability to adsorb or exclude molecules based on molecular size. However, commercial efforts with these materials could not be attempted until Milton in 1948 discovered a method for synthesizing zeolites.[5] Synthesized zeolites do not have the crystal defects of naturally occurring zeolites and could be tailored to create zeolites which were not available in nature. Molecular sieves used in oxygen concentrators and in commercial separation processes are primarily synthesized zeolites.

In 1960 Skarstrom invented the process known as pressure swing adsorption or PSA (Figure 4).[6] His discovery stemmed from having difficulties in performing a gas analysis on an air stream due to a high moisture content. He initially used the PSA process to extract moisture from the air stream (Figure 5). He also investigated the use of PSA for air separation (Figure 6).

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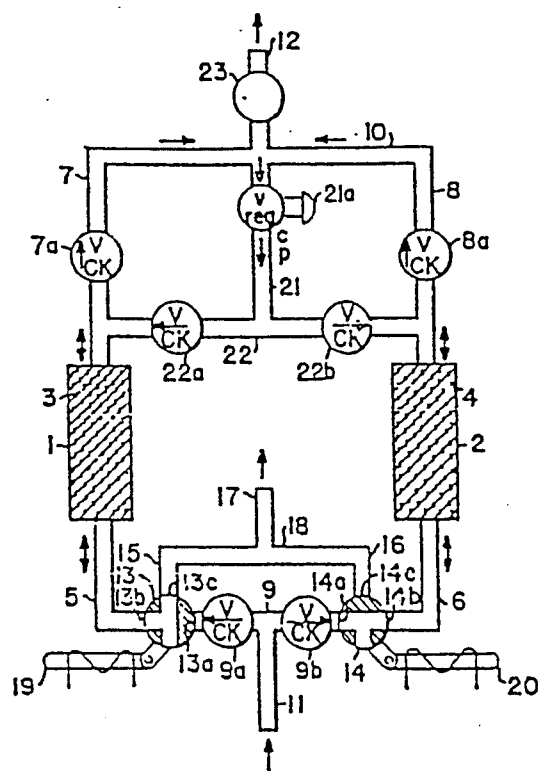
Figure 4. Skarstrom PSA Apparatus.[6]
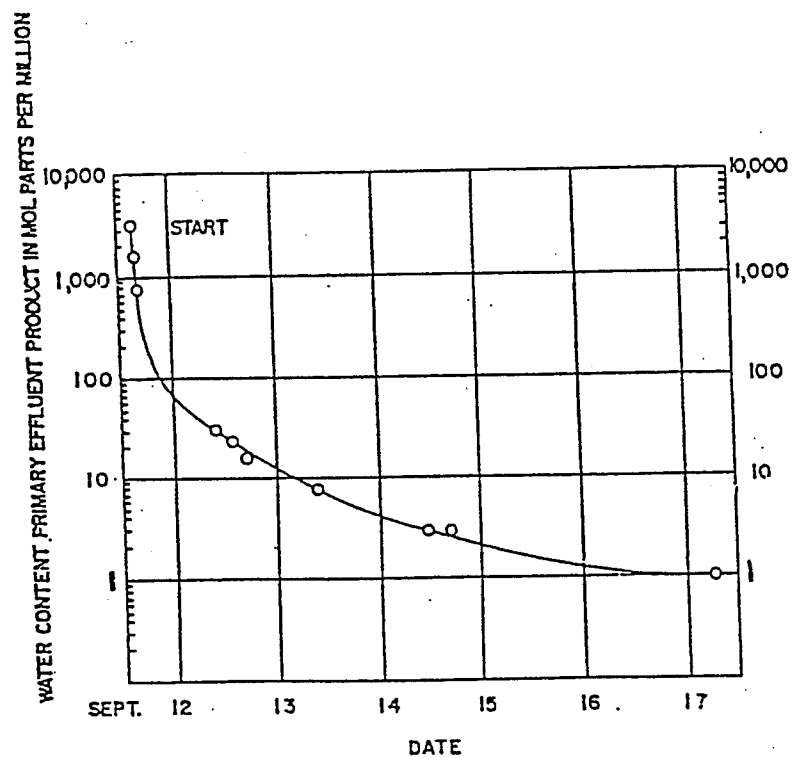
Figure 5. Water Content Data for the Skarstrom Apparatus.[6]

The first application of molecular sieves in a proposed aircraft oxygen system occurred in 1972 when a PSA process was used as a preliminary step for a barium oxide oxygen generator. This work was performed by Bendix Corporation (now Litton), Davenport, Iowa. Barium oxide was heated and exposed to oxygen to form barium dioxide. When the barium dioxide was heated to a higher temperature oxygen was released. The PSA process was used as an enrichment step to generate 50-70% oxygen from air. This oxygen was then allowed to combine with the barium oxide. The barium oxide system was not successful, however, Litton continued development of the PSA process. In 1977 Litton proposed the PSA system to the US Navy as an aircraft on-board oxygen generating system. Litton also improved the technology resulting in the production of 93-95% oxygen.
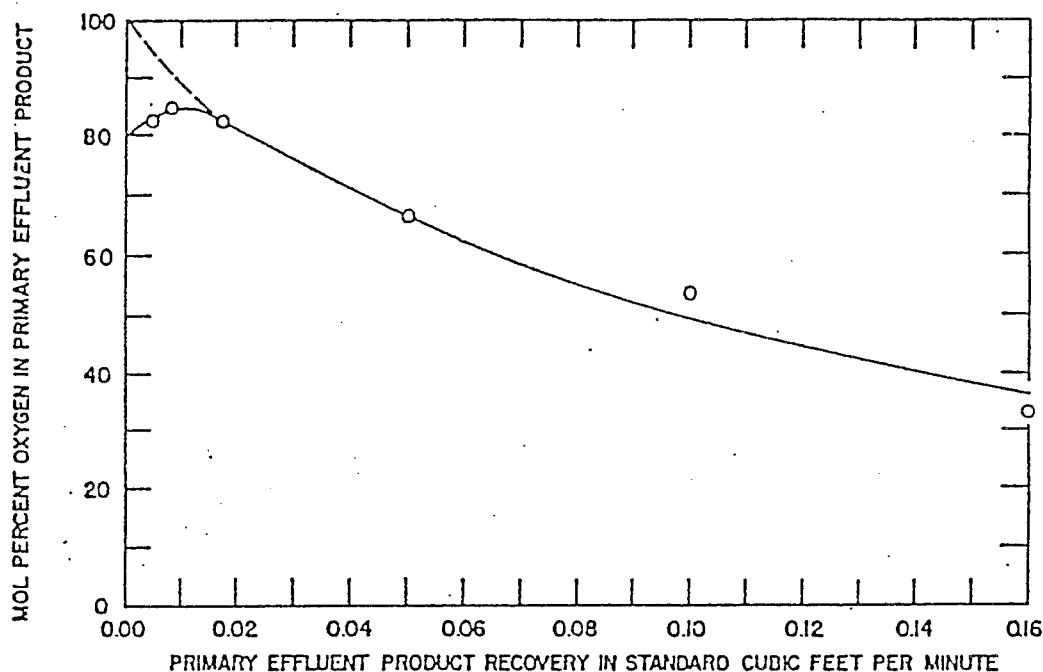
Figure 6. Oxygen Concentration Data for the Skarstrom Apparatus.[6]

Aircraft Molecular Sieve Oxygen Generating Systems

USN AV-8B Fighter In 1981 the US Navy developed the first production Molecular Sieve Oxygen Generating System (MSOGS), sometimes referred to as an On-Board Oxygen Generating System, for the AV-8B. The system was first tested on an AV-8A aircraft and then transitioned to the AV-8B.[7] The manufacturer of the system was the Bendix Corporation (now Litton), Davenport, Iowa. The MSOGS was comprised of an oxygen concentrator containing 5AMG molecular sieve, breathing regulator, oxygen monitor, and regulated emergency oxygen system (REOS) (Figures 7 and 8). The system was designed to directly replace the 5-liter liquid oxygen converter of the AV-8A aircraft. Engine bleed air supplied from the eighth stage manifold was fed to the concentrator. An inlet coalescing filter was incorporated for removal of both particulate matter and liquids including aerosol mists. An inlet air pressure reducer was used to limit air consumption. The system had a rotary inlet valve which directed bleed air to the two molecular sieve canisters. The drive motor operated on 110 VAC 400 Hertz. The concentrator cycle time (time to pressurize and depressurize one canister) was fixed at about 10 seconds. Outlet pressure fluctuations were minimized through use of a product plenum which modulated the concentrator pressure swings. The design flow rates for the system were 13.1 ALPM for the one man and 26.2 for two men, as given in MIL-D-19326E (average flow rates for the liquid oxygen system specification).
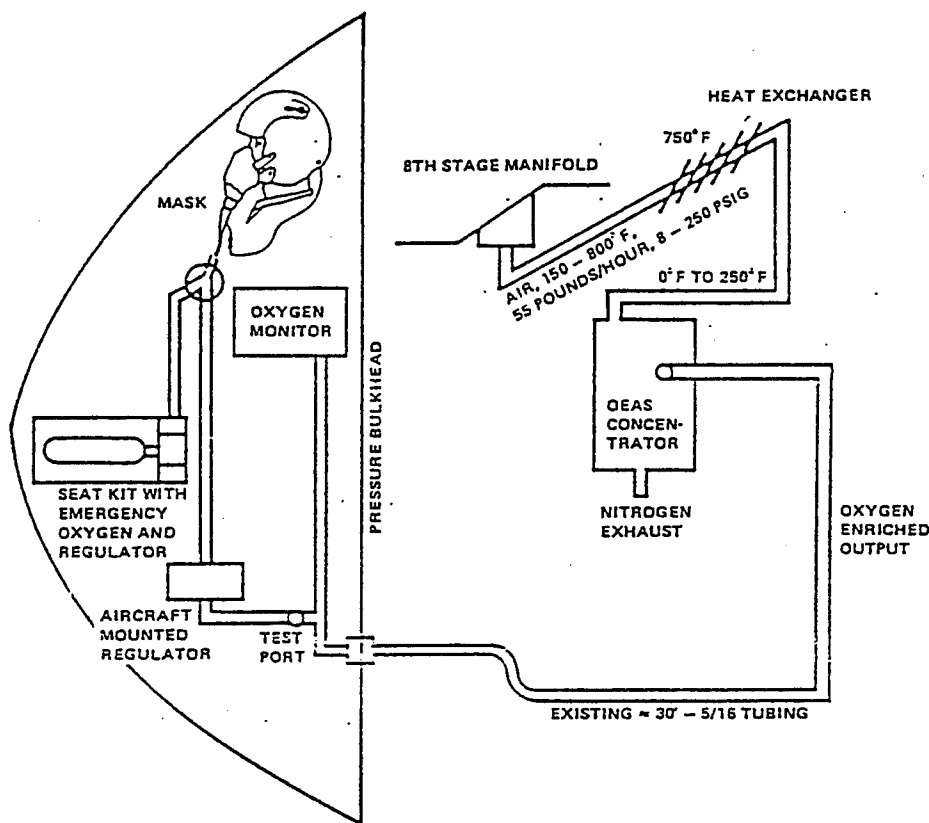
Figure 7. AV-8B Molecular Sieve Oxygen Generating System.[7]
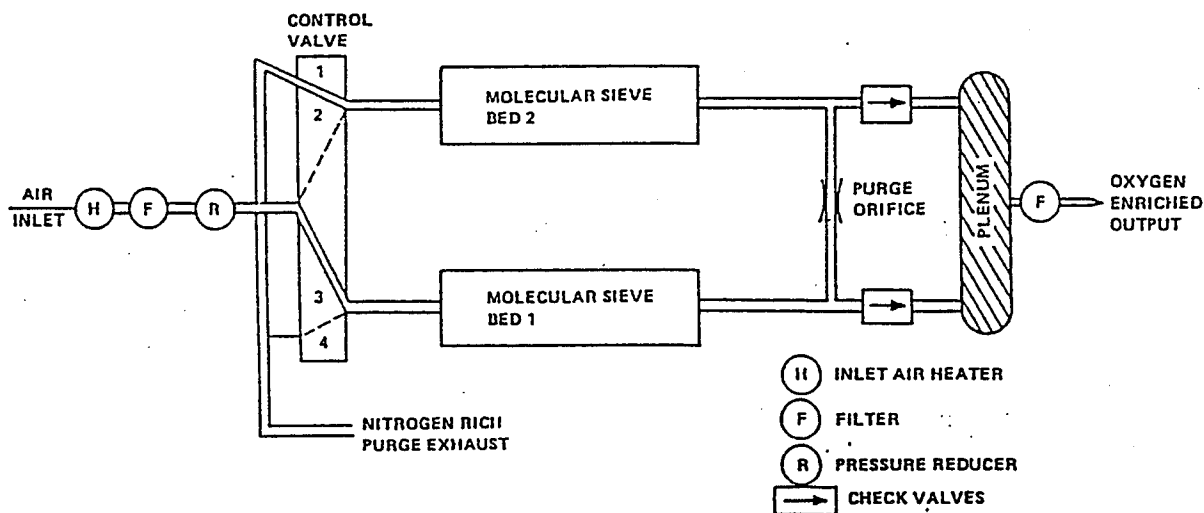
Figure 8. AV-8B Molecular Sieve Oxygen Concentrator.[7]

Typical performance curves for the system are shown in Figures 9-11. In Figure 9 the characteristic drop in performance with decreasing inlet pressure or increasing outlet flow is shown during operation at sea level. The outlet flow has units of NLPM. In Figure 10 these effects are shown for a cabin altitude of 50,000 feet. Notice the characteristic improvement in performance during operation at the higher altitude. The maximum oxygen

concentration attainable from the AV-8B system was 93-95%. In Figure 11 the effect of a higher operating temperature is shown. Generally, concentrator performance declines with increasing temperature and product flow, and decreasing inlet air pressure. During testing, problems with the control electronics and motor driven valve were corrected. The AV-8B system was qualified to an aircraft altitude of 50,000 feet. However, the current ceiling of the AV-8B is reported to be 42,000 feet. Litton recently reported that the AV-8B system had achieved a total of about 350,000 flight hours.
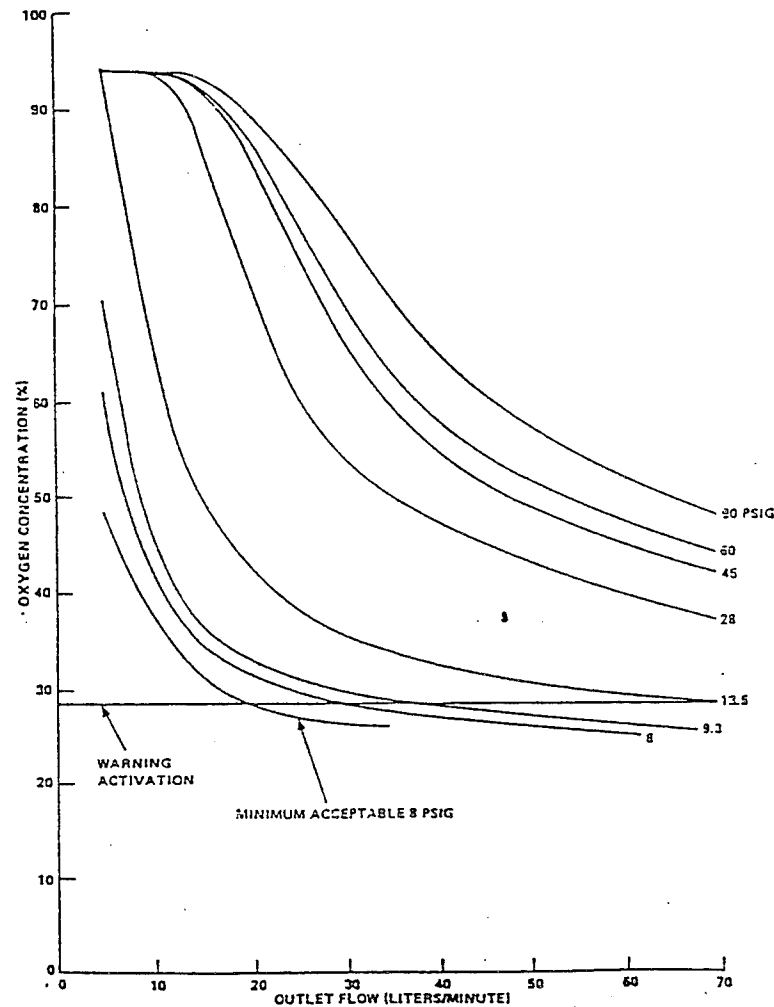
Figure 9. AV-8B MSOGS Performance at Sea Level.[7]

The concentrator gas was delivered to a low inlet pressure, pressure demand oxygen regulator, and a MBU-14/P oronasal mask. The non-dilution breathing regulator was designed to operate at the lower inlet pressures of oxygen concentrator systems. The regulator provided product gas on demand with safety pressure from ground level to 35,000 feet cabin altitude and pressure breathing between 35,000 and 42,000 feet cabin altitude. Changes in the positive pressure breathing schedule were required to maintain the alveolar oxygen partial pressure at physiologically acceptable levels due to use of 93-95% oxygen (Figure 12). Cabin differential pressure on the AV-8B was zero from ground level to 4,000 feet, varied from 0 to 3.5 psi from 4,000-30,000 feet aircraft altitude, and remained constant at 3.5 psi at aircraft altitudes from 30,000-42,000 feet.
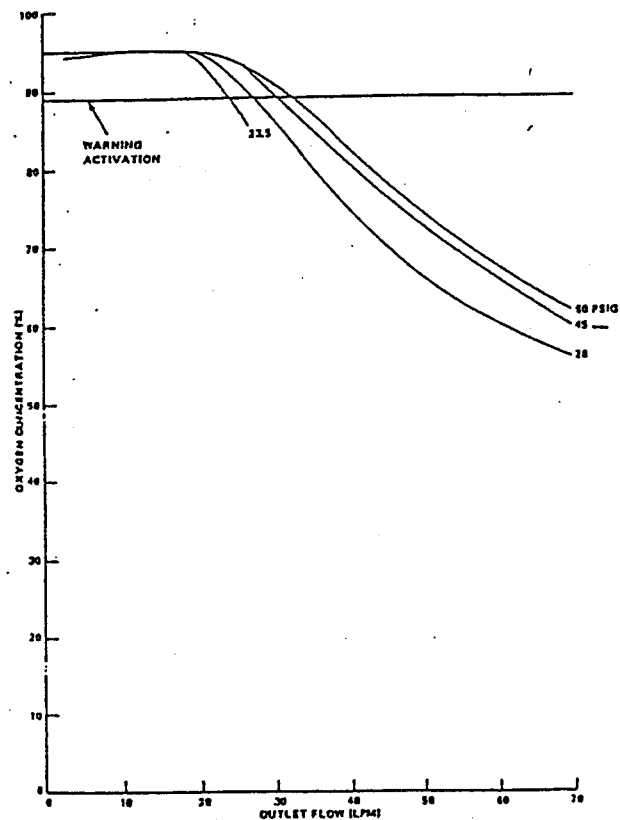
Figure 10. AV-8B MSOGS Performance at a Cabin Altitude of 50,000 Feet.[7]
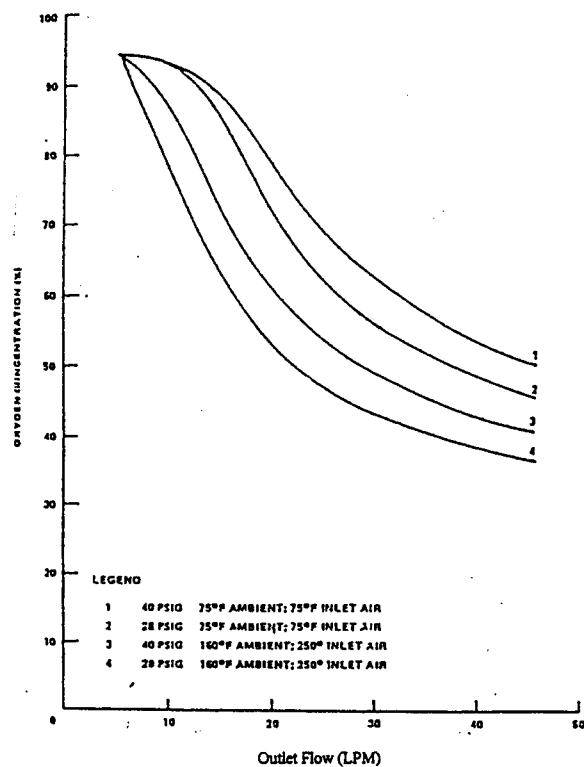
Figure 11. Effects of Temperature on AV-8B MSOGS Performance.[7]
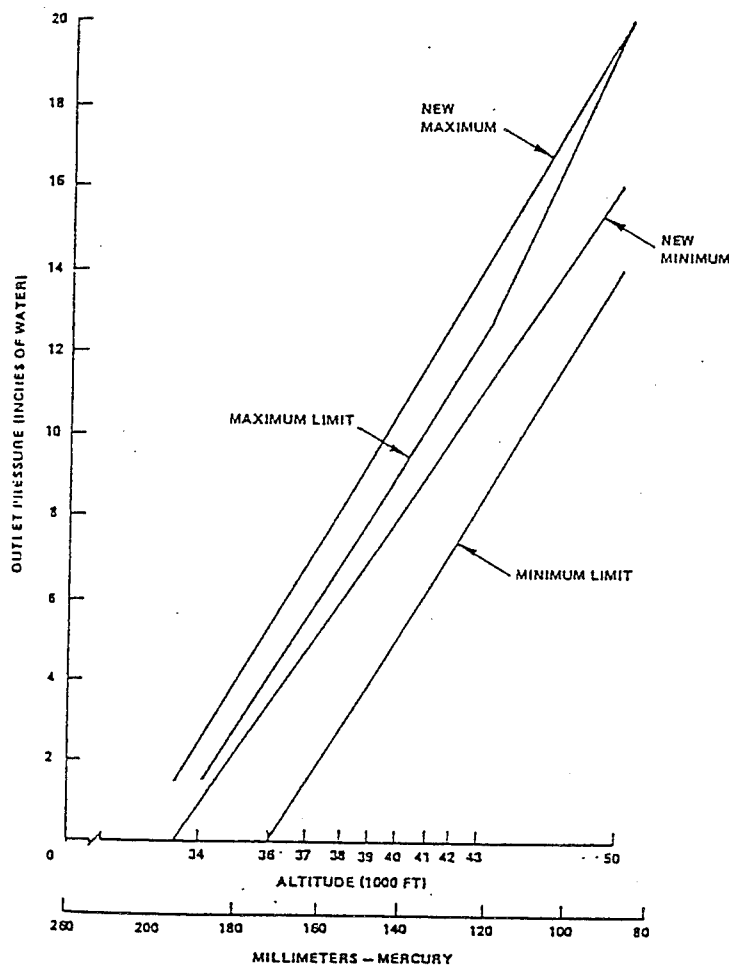
Figure 12. AV-8B MSOGS Breathing Regulator Outlet Pressure Limits.[7]

An oxygen monitor installed in the cabin was used to monitor the oxygen concentration of the gas delivered by the oxygen concentrator. The polarographic sensing element employed had a gold cathode and silver anode immersed in a gel electrolyte. The unit was sealed by an oxygen permeable membrane.

A polarizing voltage applied between these two electrodes results in a current flow which is directly proportional to the oxygen partial pressure. A warning was issued if the partial pressure of oxygen (PO_2) in the breathing gas fell below 220 ±10 mmHg (Figure 13). The AV-8B system did not have a maximum oxygen concentration specification. A press-to-test feature on the monitor supplied air to the monitor for testing the warning signal. The disadvantages of the polarographic oxygen monitor were the requirements for frequent calibration and replacement. The Navy is currently involved in a program to replace the polarographic oxygen monitor with a zirconium oxygen monitor.

The seat-mounted REOS was comprised of a high pressure oxygen bottle (1800 psig), oxygen regulator, and contents gauge, and had a capacity of 200 NL. The oxygen concentration of the gas in the bottle was 93 or 99.5% depending on what resources were available at the operating base. At remote bases the Expeditionary Oxygen and Nitrogen System (EONS) was used to fill the emergency system. The oxygen concentration delivered by EONS was 93%. The AV-8B did not have a backup oxygen system. The REOS breathing pressure can range from 2-20 in-Wg depending on the altitude. Initiation of the REOS can be accomplished manually upon receiving a warning or automatically upon ejection. Once activated the oxygen from the REOS was delivered to the crew member until the bottle was depleted.

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It's interesting to note that with the AV-8B system design a high altitude rapid decompression may or may not activate the warning signal (Figure 13). For example, the aircraft could decompress from 23,000 feet cabin altitude to 42,000 feet aircraft altitude. At this point the breathing pressure in the mask would rise to about 148.4 mm Hg (127.9 mm Hg + 20.5 mm Hg). If we assume the oxygen concentrator is delivering an average oxygen concentration of 86% (minimum for warning activation is 85%), the partial pressure of oxygen delivered to the aircrewman would be 127.6 mm Hg (0.86 x 148.4 mm Hg). In comparison, US Air Force systems automatically activate a backup oxygen system or issue an automatic warning if the cabin altitude exceeds 25,000 to 28,000 feet.
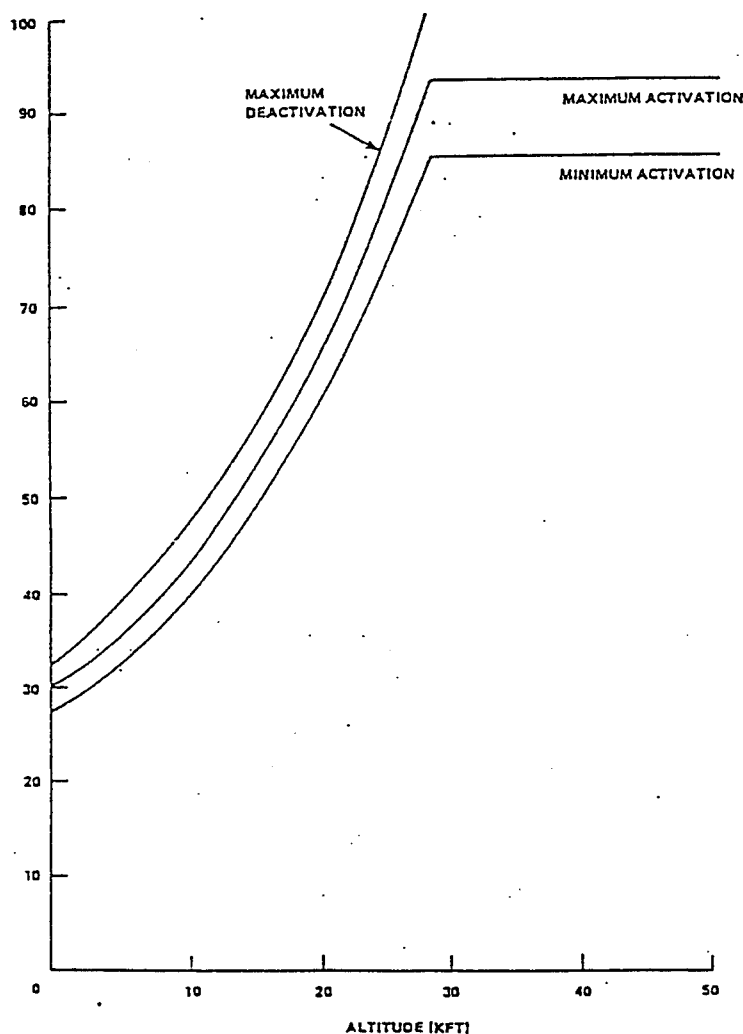
Figure 13. AV-8B Oxygen Warning Limits.[7]

USAF F-16 Prototype MSOGS In 1982 the first US Air Force MSOGS was installed on a F-16A aircraft.[8] The prototype system was comprised of an oxygen concentrator, breathing regulator, oxygen monitor, composition controller, selector valve, backup oxygen system, and emergency oxygen system (EOS) (Figure 14). The manufacturer of the system was Clifton Precision (now Litton), Davenport, Iowa.

The concentrator had two molecular sieve canisters filled with 5AMG molecular sieve and was similar to the AV-8B system. Performance curves for the system at moderate (20 ALPM) and high product flows (50 ALPM) with a pressurized and unpressurized cabin are given in Figures 15 and 16. The design flow rate for the system was 50 ALPM. The maximum oxygen concentration delivered by the system was 93-95%. The system was qualified to an aircraft altitude of 50,000 feet. This prototype system was not transitioned to the production F-16 aircraft, primarily due to funding constraints. Hence, at the present time the F-16 fleet uses a liquid oxygen system.

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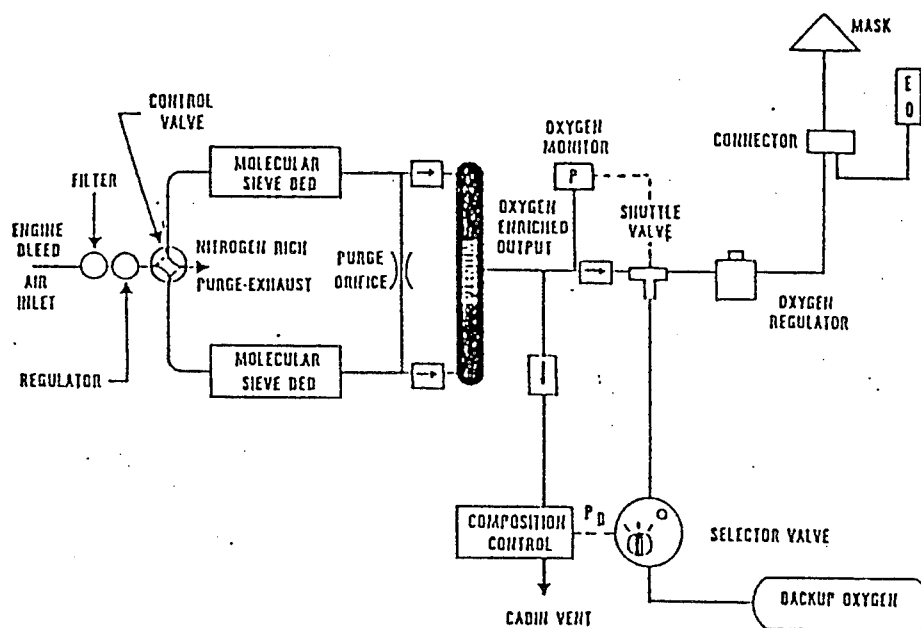
Figure 14. F-16 Prototype Molecular Sieve Oxygen Generating System.[8]
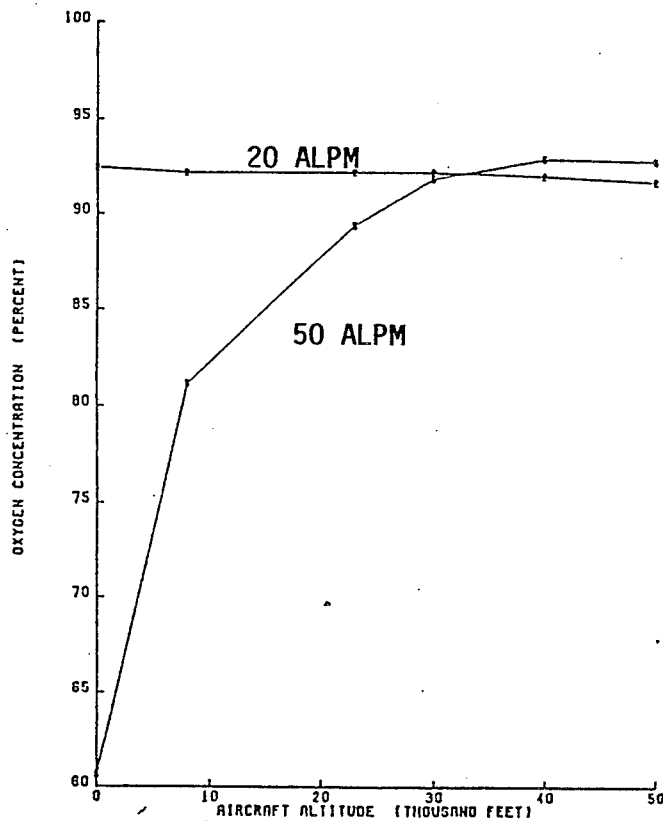
Figure 15. Performance of the F-16 MSOGS at Moderate and High Flow Rates During Pressurized Flight at 23°C Inlet Temperature and 40 psig Inlet Pressure (2 = 20 ALPM, 5 = 50 ALPM).[8]
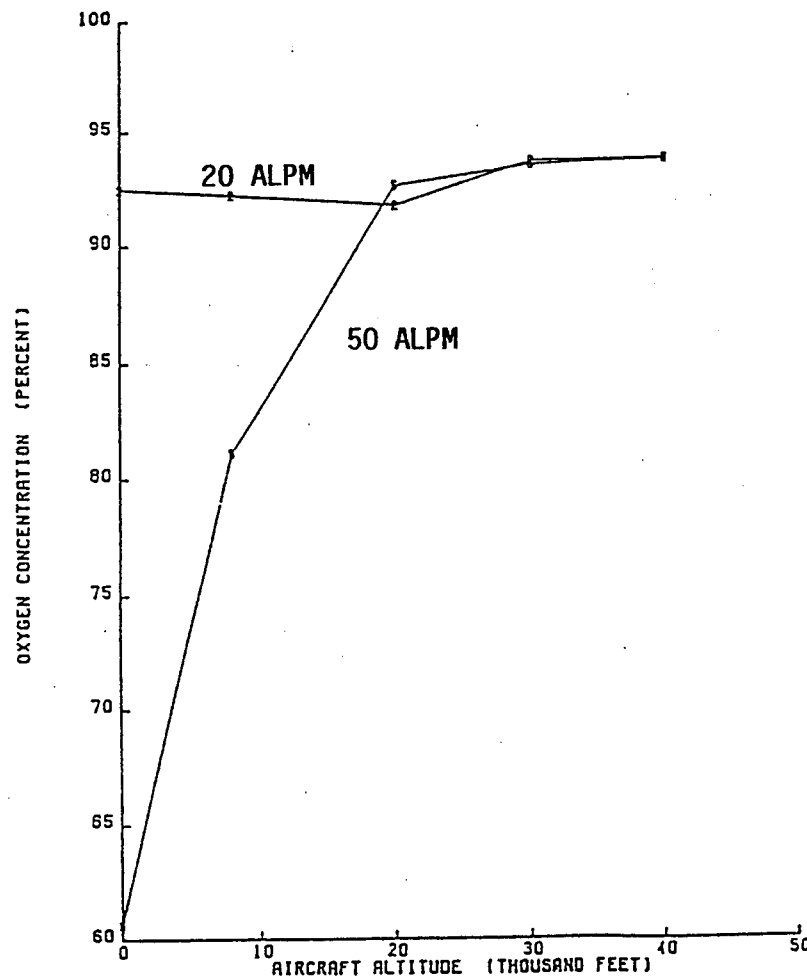
Figure 16. Performance of the F-16 MSOGS at Moderate and High Flow Rates During Unpressurized Flight at 23°C Inlet Temperature and 40 psig Inlet Pressure (2 = 20 ALPM, 5 = 50 ALPM).[8]

The F-16 MSOGS was one of the first units to incorporate an oxygen composition controller to adjust the product gas oxygen concentration. This controller simply vented additional product gas into the cabin, thereby increasing the product flow from the concentrator and lowering its oxygen composition. A composition controller was required because the system had a minimum and maximum oxygen specification (Figure 17). The US Air Force specified that the product gas oxygen concentration should be ≤70% from ground level to 17,000 feet cabin altitude. This requirement was included to eliminate the occurrence of acceleration atelectasis. The F-16 cockpit is unpressurized from ground level to 8,000 feet, remains at 8,000 feet from an aircraft altitude of 8,000 feet to 23,000 feet, and maintains a differential pressure of 5 psid from 23,000 feet to 50,000 feet aircraft altitude (Figure 18).

The system had a pressure demand, non-dilution breathing regulator. The regulator delivered 1 in-Wg positive pressure from ground level to 38,000 feet, where pressure breathing began. The regulator was designed to provide pressure breathing for altitude protection up to 50,000 feet. The regulator pressure breathing schedule is given in Figure 19.

The F-16 MSOGS had a polarographic oxygen monitor similar to the AV-8B monitor. This monitor provided a low oxygen warning to the pilot and automatically activated the BOS, if the concentrator was producing an insufficient oxygen partial pressure. The monitor issued a warning if the oxygen partial pressure fell below 195 mm Hg. Problems with the F-16 oxygen monitor were similar to problems with the AV-8B monitor, mainly frequent calibration and replacement.

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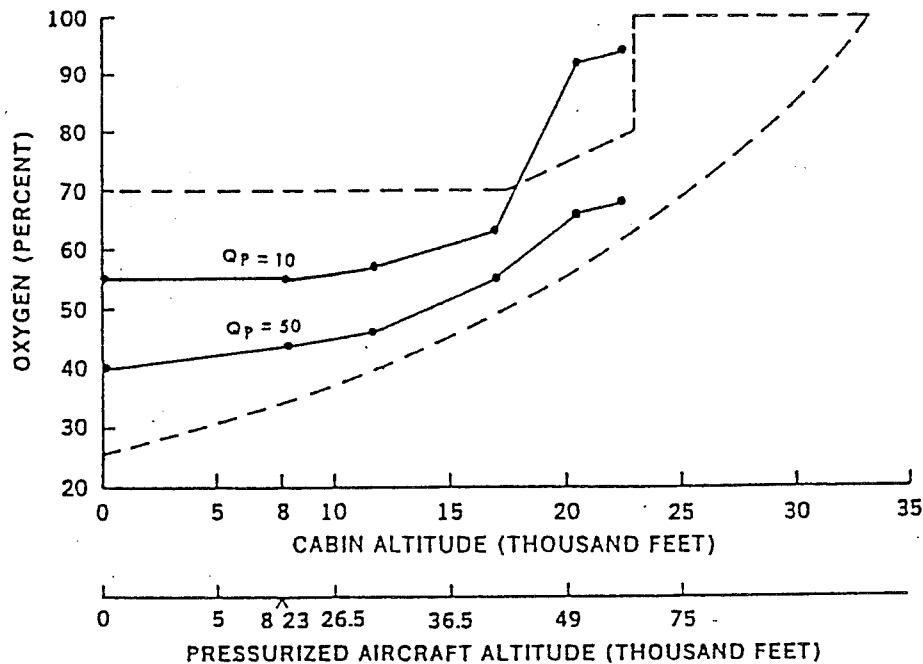
Figure 17. Minimum and Maximum Oxygen Specification and Oxygen Concentrator Performance for the F-16 MSOGS at Product Flows of 10 and 50 ALPM, Inlet Pressure of 40 psig, and Inlet Temperature of 80°C.[8]
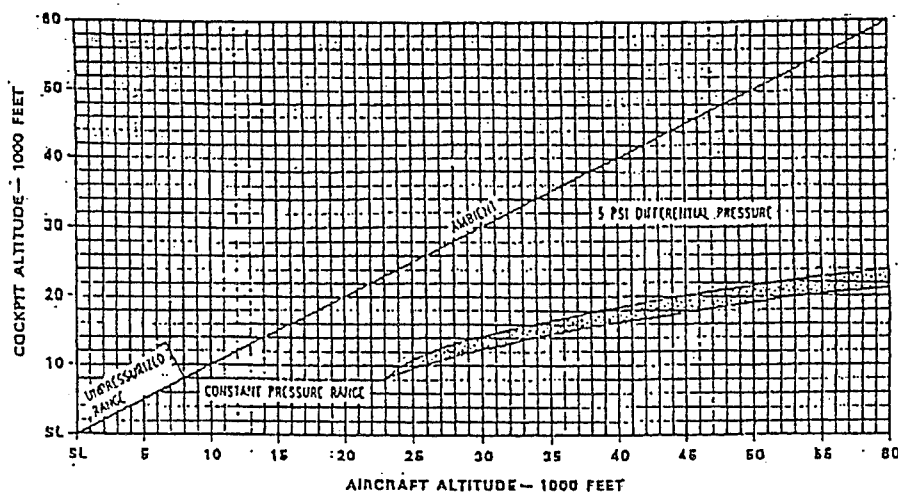
Figure 18. F-16 Cockpit Pressurization Schedule.

The backup oxygen system (BOS) consisted of two 50 in^3 high pressure (1800 psig) gaseous oxygen cylinders having a combined capacity of 200 NL. With the system in the automatic mode, the BOS activated automatically if the cabin altitude exceeded 25,000 feet, breathing gas oxygen partial pressure fell below 195 mm Hg, or system pressure dropped to 10 psig or below. A high pressure hose connected the BOS bottles to the selector valve which reduced the pressure to 60 psig and delivered the backup oxygen to the regulator.

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USAF B-1B Bomber In 1985 the USAF completed development of the B-1B MSOGS.[9] The unit was comprised of a concentrator assembly, a release valve, two purge valves, and six breathing regulators (Figure 20). The normal and extended crew size was four and six, respectively. The system was manufactured by Normalair-Garrett Ltd., Yeovil, United Kingdom. The MSOGS was designed for an outlet flow rate of 160 ALPM (26.7 ALPM per crew member for a six man crew). The B-1B MSOGS had six molecular sieve canisters filled with MG3 molecular sieve. (The MG3 molecular sieve was later replaced with OXYSIV-5 molecular sieve.[10]) The MSOGS was supplied engine bleed air which was cooled, dehumidified, filtered, and pressure regulated to 32 psig. A centrifugal water separator was used to extract liquid water from the bleed air exiting the concentrator assembly heat exchanger. The cycle time of the concentrator was fixed at about 9 seconds. In Figure 21 the system performance curves are shown for both MG3 and OXYSIV-5 molecular sieves under nominal operating conditions. System performance is considerably higher with OXYSIV-5 molecular sieve. In Figure 22 system performance is shown for worst case operating conditions (high temperature and low pressure) with an unpressurized cabin. The system did not have a maximum oxygen concentration specification. Further, the system did not have an oxygen composition controller or oxygen monitor. The B-1B MSOGS was qualified to an aircraft altitude of 42,000 feet.
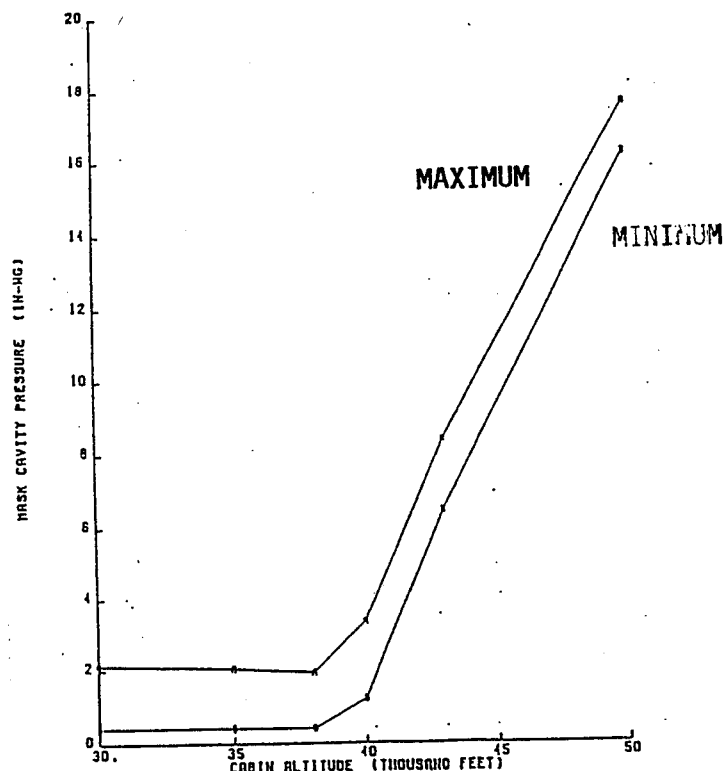
Figure 19. F-16 MSOGS Regulator Pressure Breathing Schedule for Altitude.[8]

A non-dilution, pressure demand regulator delivered MSOGS breathing gas to the aircrew mask. The non-dilution capability ensured that chemical contaminants would not be introduced into the regulator through a dilution port. The minimum operating pressure for the regulator was 10 psig. The regulator incorporates an anti-suffocation valve, a compensated outlet relief valve, and a NORM/PRESS mode selector for switching from demand to safety pressure breathing. The regulator was ejection seat-mounted for the four primary crew members and man-mounted for the two instructors. Automatic pressure breathing began at a cabin altitude of 30,000 feet which increases to 8-10 in-Wg (15-19 mm Hg) at 45,000 feet. A safety pressure of about 1.5 in-Wg (3 mm Hg) can be manually selected. Final delivery of the breathing gas is through a Type MBU-12/P oronasal mask.

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LEGEND

HX = HEAT EXCHANGER (PRE-COOLER)
F = FILTER
⊗ = MANUAL SHUT-OFF
---▶ = ELECTRICAL SIGNAL
∿ = FLEXIBLE HOSE

WS = WATER SEPARATOR
Z = ZEOLITE BED
BOS = BACK-UP OXYGEN SUPPLY
CITS = CENTRAL INTEGRATED TEST SYSTEM

R = REGULATING VALVE
ECU = ELECTRONIC CONTROL UNIT
EO = EMERGENCY OXYGEN
►─ = CHECK VALVE
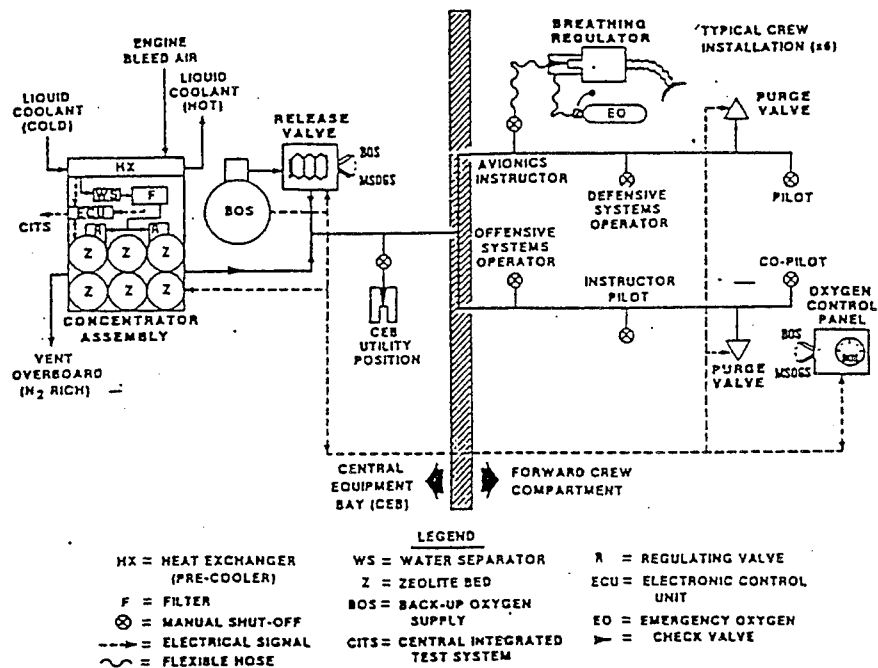
Figure 20. B-1B Molecular Sieve Oxygen Generating System.[9]
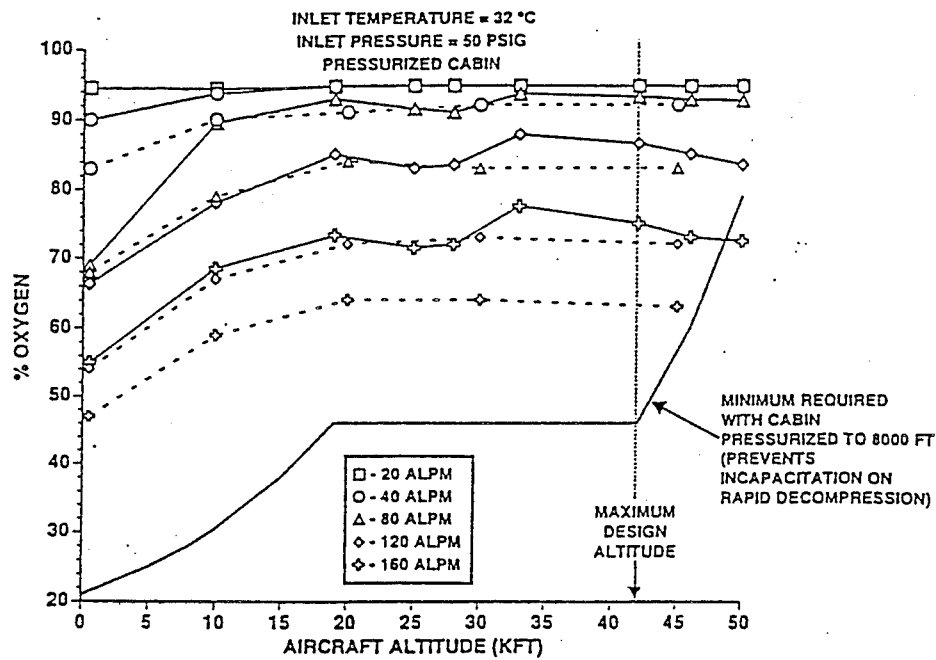
Figure 21. Performance Curves for the B-1B MSOGS with OXYSIV-5 and MG3 Molecular Sieve During Pressurized Flight (--- MG3, __ OXYSIV-5).[10]

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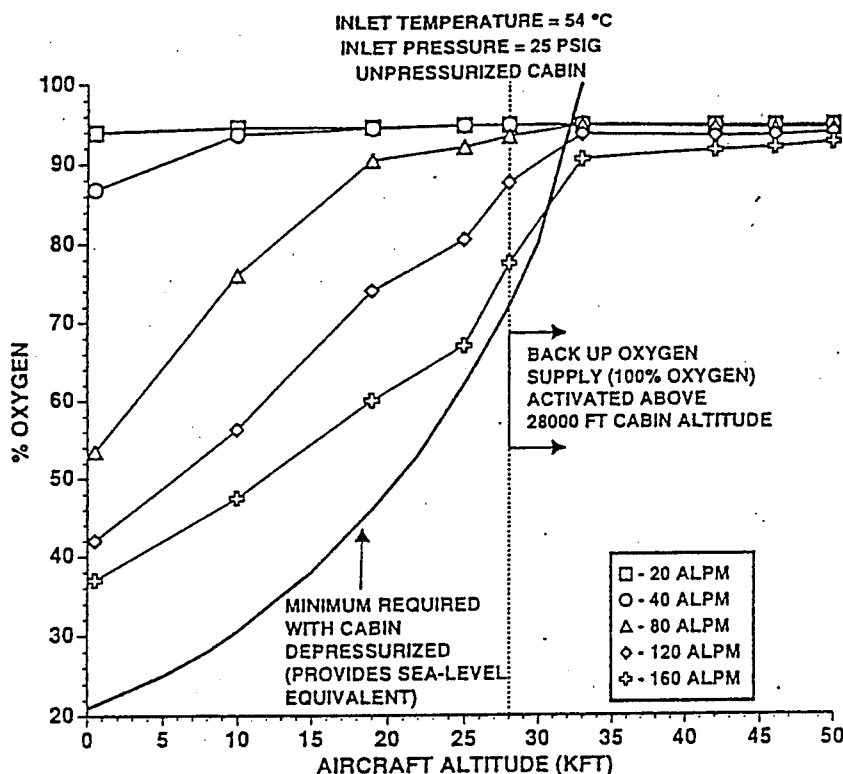
Figure 22. Performance Curves for the B-1B MSOGS with OXYSIV-5 Molecular Sieve During Unpressurized Flight.[10]

An aircraft mounted backup oxygen system contained 2,800 NL of 99.5% oxygen. The BOS was automatically activated if the cabin altitude exceeded 28,000 feet. Also, the BOS could be manually selected by the aircrew. The aircrew might select the BOS in case of problems with the aircraft bleed air supply, MSOGS concentrator failure, and smoke or fumes in either the cabin air supply or the MSOGS product gas, or if the aircrew experienced airsickness, hypoxic symptoms, or any other physiologic distress. Also, each crew member had the standard seat-mounted emergency oxygen system containing 50 NL.

After the B-1B MSOGS had been fielded for about two years problems with molecular sieve "dusting" developed. Dusting occurs when the molecular sieve pellets breakdown due to their unrestricted movement within the canisters. The molecular sieve dust then flows out of the system with the product and exhaust gases. It has been speculated that dusting on the B-1B system may occur because of a loss of bed retention and/or exposure to liquid water. If molecular sieve pellets are not properly retained in the canister during the pressurization cycles, they will migrate and begin the process of attrition which eventually leads to dusting. If liquid water should enter the molecular sieve canisters, the high heat of adsorption which will occur when water adsorbs within the molecular sieve could also lead to pellet attrition. In the future the molecular sieve in the B-1B system will be immobilized with a polymer. The polymer will bind together the molecular sieve pellets to prevent "dusting." Presently, any dust produced by the MSOGS is captured by a downstream particulate filter (not shown in Figure 20). This outlet particulate filter was added after the system was fielded. The B-1B MSOGS design attempts to condition raw engine bleed air which can have an extremely high moisture content during low level flight. Most other MSOGS receive engine bleed air which has been processed by the aircraft environmental control system.

The B-1B MSOGS typically delivers higher oxygen concentrations than desired due to the system size (sized for a crew of six) and the lack of a composition controller. High oxygen concentrations during extended missions have resulted in cases of delayed otic barotrauma. The US Air Force has considered the incorporation of a composition controller and an oxygen monitor for the B-1B system.

USAF Tactical Life Support System (TLSS) In 1986 a prototype MSOGS flew on a F-15B aircraft.[11,12] The system was manufactured by Normalair-Garrett Ltd., Yeovil, United Kingdom. The oxygen concentrator had three molecular sieve beds containing MG3 molecular sieve. The design flow rate for the two crew member system was 120 ALPM. Performance curves for the system at nominal operating conditions are shown in Figure 23. Oxygen concentration was controlled by cycling the canisters at a fast or a slow speed. The fast cycle time permitted an increase in the oxygen concentration while the slow cycle time allowed the nitrogen concentration wavefront to further penetrate the molecular sieve resulting in a lower oxygen concentration. The system limited the maximum oxygen concentration to ≤65% at cabin altitudes below 12,500 feet. A fluidic oxygen sensor was used to monitor the outlet oxygen concentration. The sensor compared the pressure drop of a reference gas (bleed air) and the product oxygen flow through capillary tubes to determine the oxygen concentration. The TLSS system was qualified to an aircraft altitude of 60,000 feet.

The TLLS system had a non-dilution regulator with pressure breathing for G capability (Figure 24). Get-me-down capability from 60,000 feet was possible by positive pressure breathing (PPB) up to 70 mm Hg at the breathing mask with an equal pressure applied to a chest counterpressure garment (or jerkin) (Figures 25 and 26). For example, on a rapid decompression from 22,500 feet cabin altitude to 60,000 feet aircraft altitude, the partial pressure of oxygen delivered by the system would be about 124.1 mm Hg (54.1 + 70 mm Hg). The oxygen concentration delivered would be 99.5%. This calculation neglects the potential occurrence of aerodynamic suction which could increase the cabin altitude. The lower G garment was supplied with four times the breathing pressure.

The aircraft mounted backup oxygen system contained 190 NL of 99.5% oxygen. The BOS automatically activated if the cabin altitude exceeded 26,000 feet. At reduced inlet pressures or oxygen concentrator failure below 9,000 feet the system switched to cabin air and illuminated the cabin air light on the regulator. At reduced inlet pressures or oxygen concentrator failure above 9,000 feet, the system switched to BOS and illuminated the 100% oxygen light on the regulator. The TLLS seat-mounted emergency oxygen system had a capacity of 100 NL.
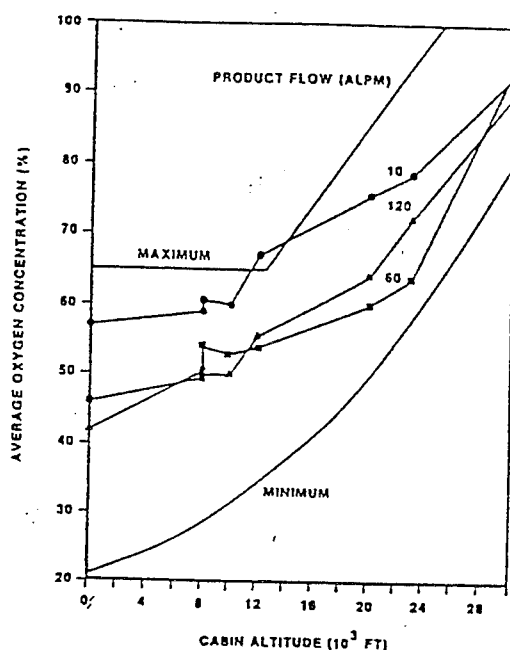
Figure 23. Performance Curves for the TLSS Molecular Sieve Oxygen Generating System at an Inlet Pressure of 40 psig and Inlet Temperature of 22°C (cabin/aircraft altitude: GL/GL, 8K/8K, 8K/23K, 10K/10K, 12K/30K, 20K/20K, 23K/60K, 30K/30K).[11, 12]

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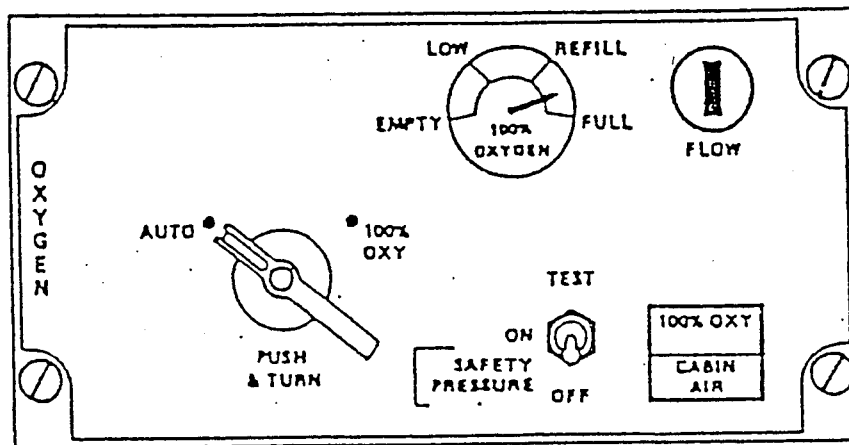
Figure 24. TLSS Breathing Regulator.[11]

USAF Oxygen Generation and Distribution System (OGADS) In 1988 the USAF completed development of the OGADS.[13] The system was manufactured by Normalair-Garrett Ltd., Yeovil, United Kingdom. The oxygen concentrator had three molecular sieve beds filled with MG3 molecular sieve. Control of the oxygen concentration was achieved by operating the system at two cycle speeds (a fast and a slow speed). The design flow rate for the system was 120 ALPM. Crew size was two or three. Typical performance curves are shown in Figures 27 and 28. The system was unable to control the oxygen concentration within predetermined ranges. Like the B-1B system, the OGADS system did not have a maximum oxygen concentration specification. A fluidic oxygen monitor was used to monitor the oxygen concentration. The system had a non-dilution breathing regulator. The OGADS system was qualified to an aircraft altitude of 50,000 feet.
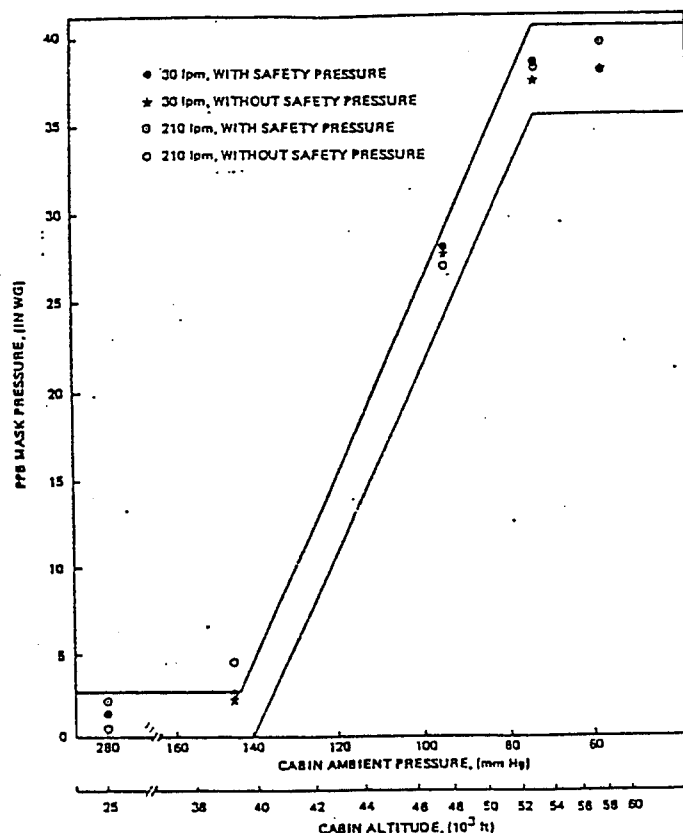
Figure 25. TLSS Pressure Breathing Schedule for Altitude.[11]

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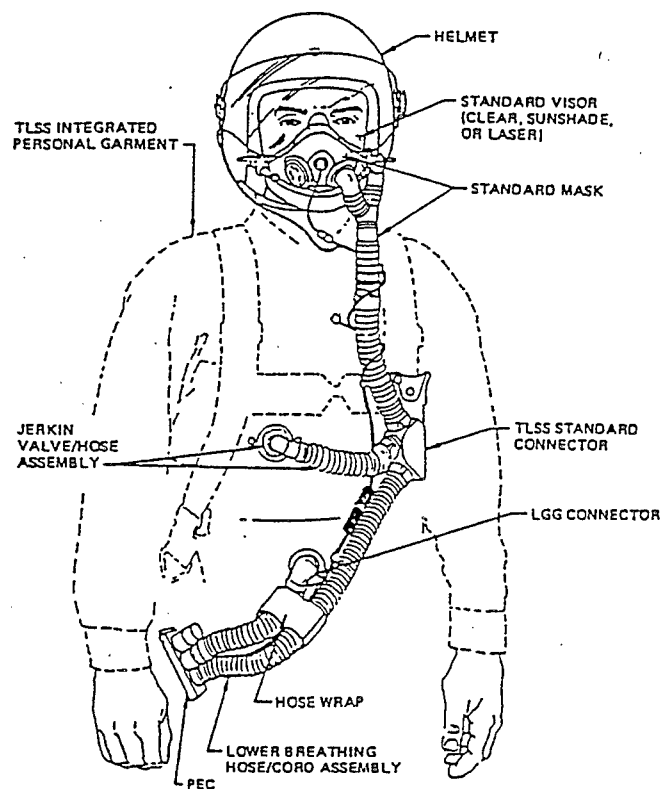
Figure 26. TLSS Standard Ensemble.[11]

The OGADS system was equipped with both standard and high altitude breathing gear. The high altitude ensemble consisted of a Modified Life Support System (MLSS) with jerkin assisted positive pressure breathing, lower body counterpressure garment, and the TLSS mask and helmet. The high altitude ensemble was designed to provide emergency protection in the range from 40,000 to 60,000 feet. Maximum mask pressure was 70 mm Hg. The lower pressure garment was pressurized to four times the breathing pressure. Problems with the high altitude gear limited the qualification of the system to an aircraft altitude of 50,000 feet. The standard ensemble consisted of a MBU-12/P oxygen mask and HGU-55/P flight helmet. The maximum mask pressure for the standard ensemble was 30 mm Hg. The standard ensemble was tested up to 50,000 feet for get-me-down capability.
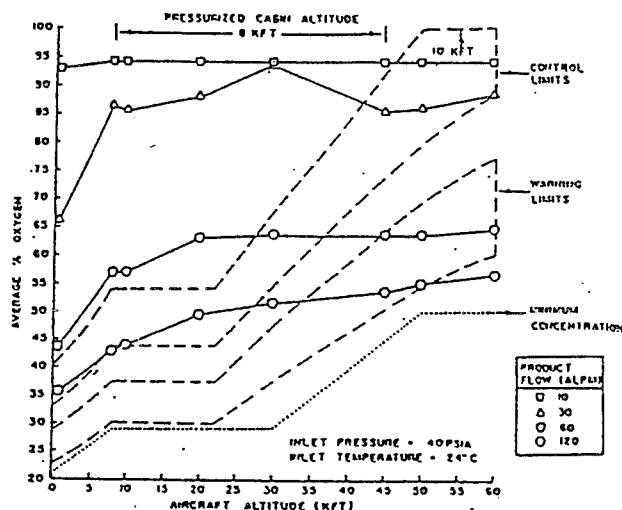
Figure 27. Performance Curves for the OGADS with a Pressurized Cabin.[13]

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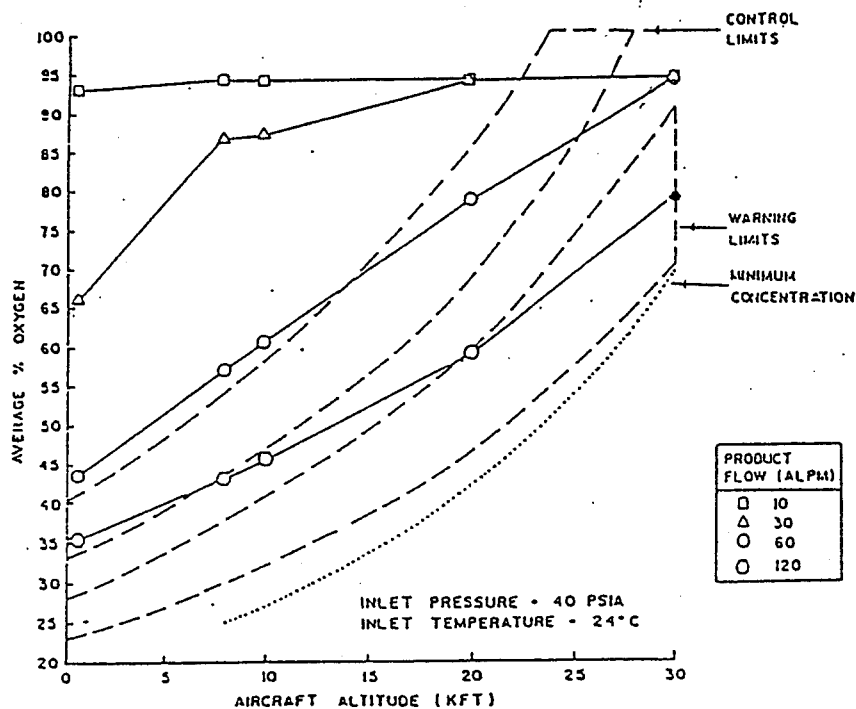
Figure 28. Performance Curves for the OGADS with an Unpressurized Cabin.[13]

OGADS had a backup oxygen system with a capacity of 850 NL. BOS activation occurred automatically at cabin altitudes of 28,000 feet and above. Also, the BOS could be manually activated by the aircrew. A seat-mounted emergency oxygen system with a capacity of 50 NL was available for each crew member.

USAF F-15E Fighter In 1991 the USAF completed development of the F-15E MSOGS.[14] The MSOGS was comprised of an oxygen concentrator with an integral self-charging backup oxygen system, zirconium oxygen monitor, and two CRU-98 breathing regulators (Figure 29). The system was manufactured by Litton, Davenport, Iowa. The oxygen concentrator was designed for a two man aircrew and a total flow rate of 100 ALPM. The two molecular sieve beds were filled with OXYSIV-5 molecular sieve. The cycle time of the concentrator was fixed at one speed (about 10 seconds). Performance curves for the F-15E system are shown in Figures 30-33 for nominal operating conditions. At the low product flow (10 ALPM) in the NORMAL mode the system was not able to maintain the oxygen concentration within the aircraft oxygen schedule (Figure 30). However, this consequence was not a major discrepancy because the outlet flow would typically be above 10 ALPM. The MSOGS had many built-in-test features to monitor system operation. The F-15E MSOGS was qualified to an aircraft altitude of 50,000 feet. Further, the system was qualified with the Combined Advanced Technology Enhanced G Ensemble (COMBAT EDGE).

An integrated zirconium oxygen monitor sensed the oxygen concentration in the product gas. The monitor operates by exposing a zirconium wafer to a reference gas (bleed air) on one side and a sample gas (product oxygen) on the other side. The wafer generates a voltage which can be related to the concentration of oxygen in the sample gas. The wafer must be heated to about 800°C for proper conduction of the oxygen ions. Presently, zirconium oxygen monitors appear to have the stability and reliability for airborne use.

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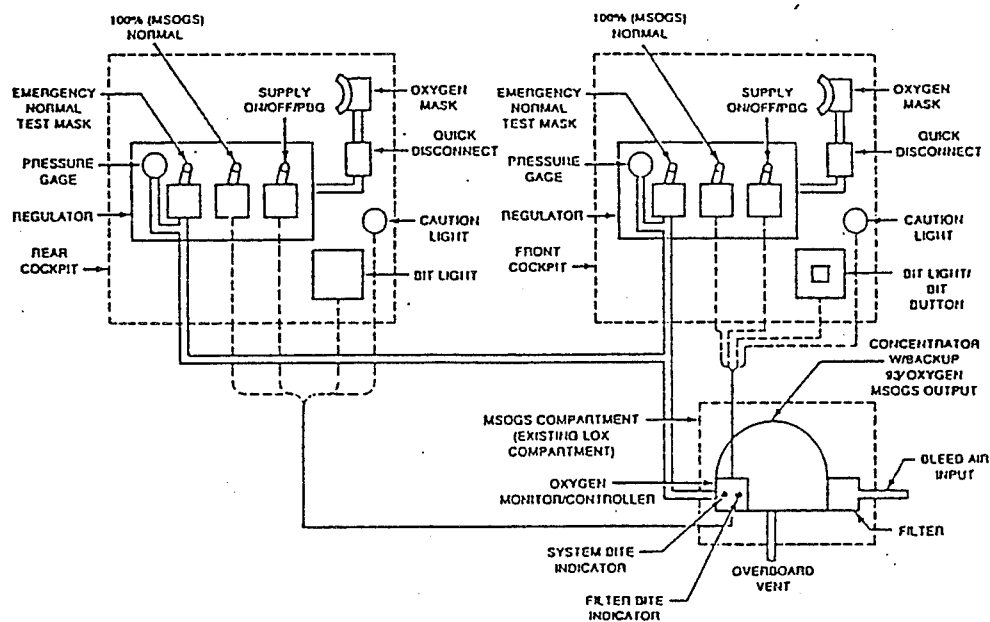
Figure 29. Simplified Schematic of F-15E MSOGS.
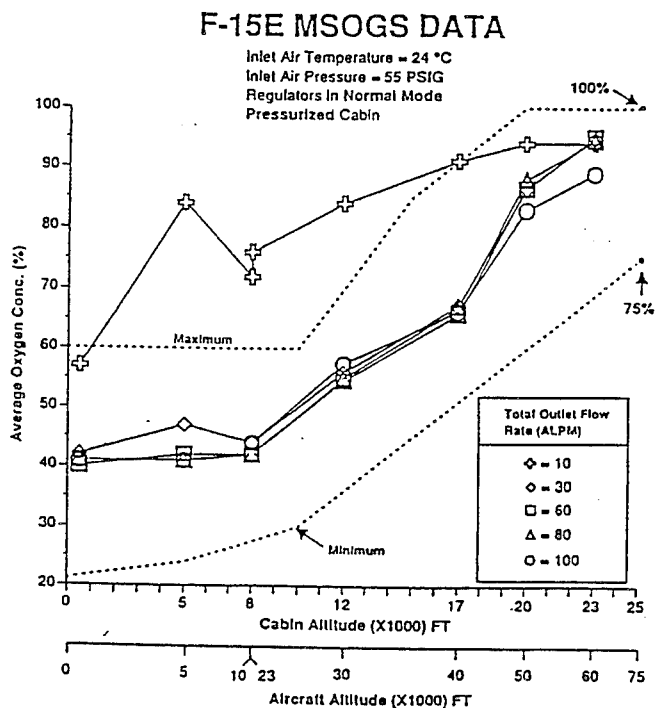
Figure 30. F-15E MSOGS Performance Curves in Normal Mode with a Pressurized Cabin.[14]

F-15E MSOGS DATA

Inlet Air Temperature = 24 °C
Inlet Air Pressure = 55 PSIG
Regulators in Normal Mode
Unpressurized Cabin
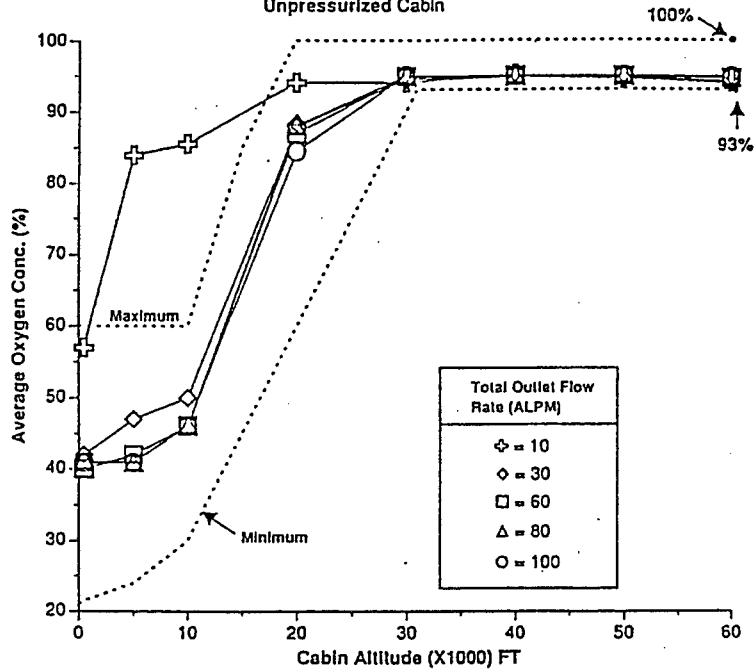
Figure 31. F-15E MSOGS Performance Curves in Normal Mode with an Unpressurized Cabin.[14]

F-15E MSOGS DATA

Inlet Air Temperature = 24 °C
Inlet Air Pressure = 55 PSIG
Regulators in 100% Mode
Pressurized Cabin
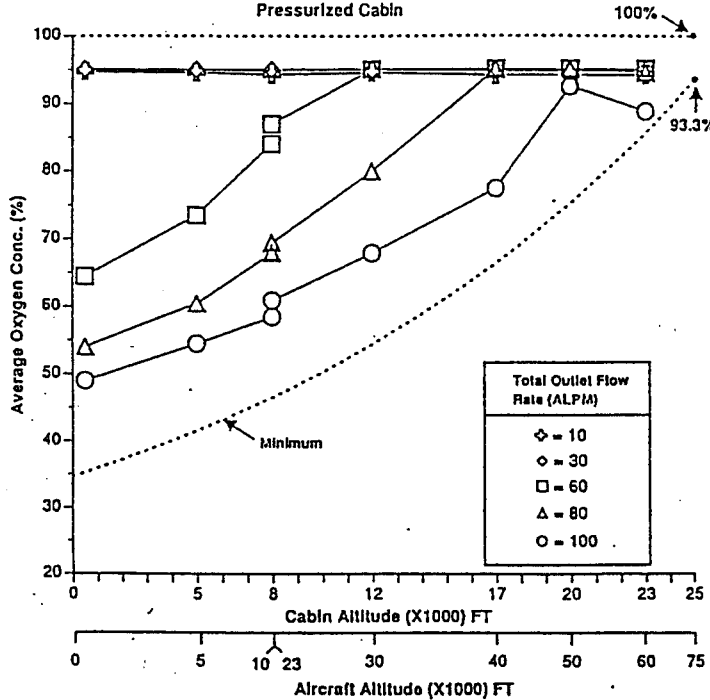
Figure 32. F-15E MSOGS Performance Curves in 100% Mode with a Pressurized Cabin.[14]

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The MSOGS CRU-98 breathing regulator is a panel mounted, diluter demand, G-compensated, oxygen regulator similar to the standard CRU-73 currently being used on the previous F-15 aircraft (Figure 34). The pressure breathing with altitude schedule is shown in Figure 35. Modifications were made to provide positive pressure breathing as a function of G forces (PBG). This function is activated by a pressure signal from an external anti-G valve. In the NORMAL mode concentrator product gas is diluted with cabin air. In the 100% mode undiluted concentrator product gas with a minimum oxygen partial pressure of 263 mm Hg is delivered to the aircrew.
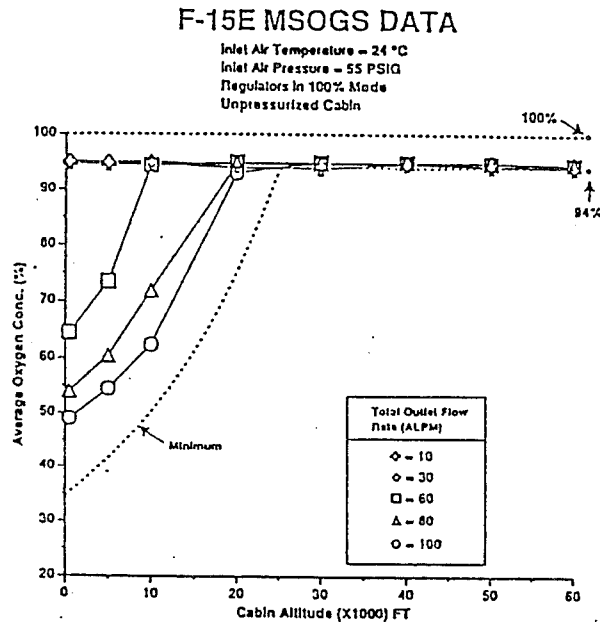
Figure 33. F-15E MSOGS Performance Curves in 100% Mode with an Unpressurized Cabin.[14]
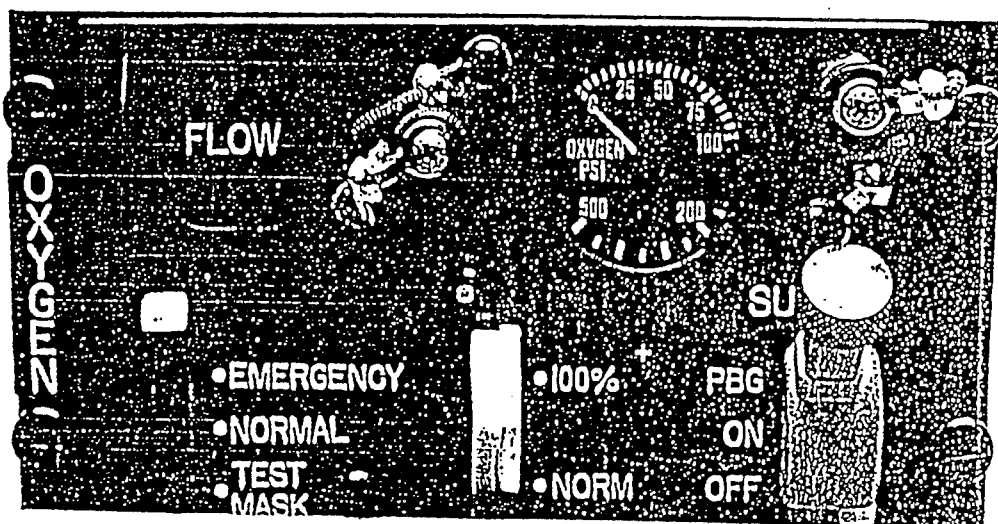
Figure 34. F-15E MSOGS Breathing Regulator.[14]

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The F-15E MSOGS had a self-charging 93% backup oxygen system which only charged when the oxygen concentrator produced ≥93% oxygen. Product oxygen was compressed to 450 psig by an integral dual piston compressor operated by engine bleed air. The BOS storage plenum contained 5AMG molecular sieve which allowed the storage of greater quantities of oxygen when compared to a standard plenum.

This system provided 10 minutes of breathing gas for the two man aircrew on the ground with the engines off. BOS capacity was about 260 NL. The BOS activated automatically if the zirconium oxygen monitor measured an oxygen concentration below the minimum oxygen specification. During rapid decompression testing the BOS delivered ≥93% oxygen to the mask within two to three breaths (Figure 36). Each crew member had an emergency oxygen system with a capacity of 50 NL of 99.5% oxygen.
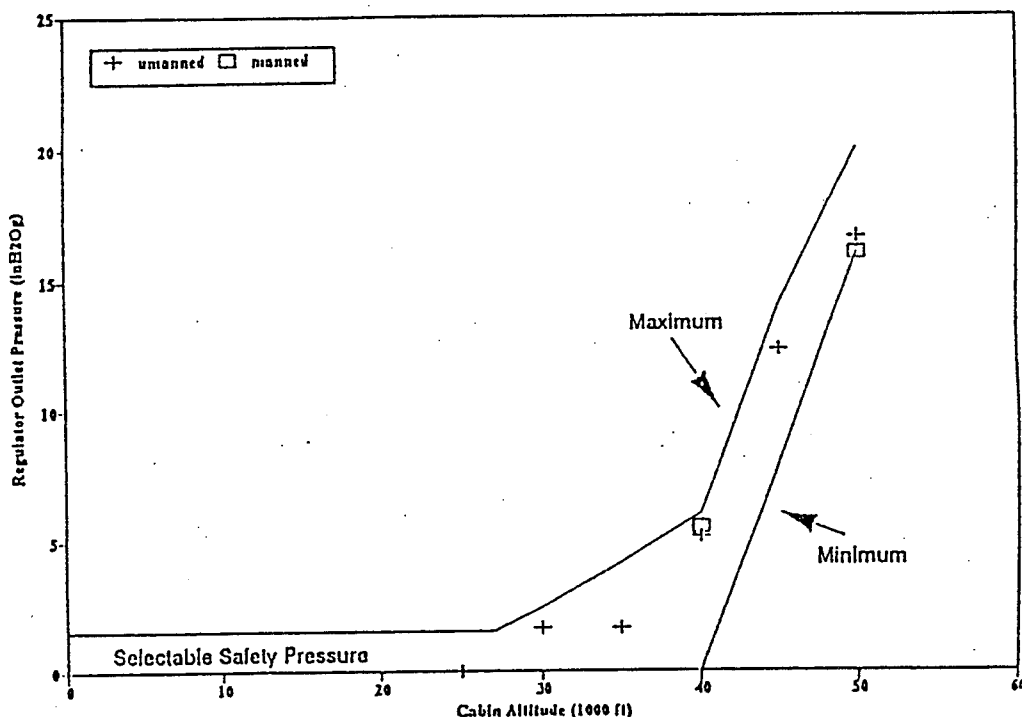
Figure 35. F-15E MSOGS Pressure Breathing Schedule for Altitude.[14]

USAF F-22 Fighter Presently, the USAF is developing the F-22 MSOGS. The critical design review for the system was completed in 1994. The system will be manufactured by Normalair-Garrett Ltd., Yeovil, United Kingdom. The oxygen concentrator will have three molecular sieve canisters loaded with immobilized OXYSIV-5 molecular sieve. Hence, molecular sieve dusting should not be a problem with this design. A zirconium oxygen sensor will monitor the oxygen concentration of the product gas. Design flows are 60 ALPM for the one man system and 120 ALPM for the two man system. The oxygen schedule for the F-22 MSOGS is shown in Figure 37. The warning band reflects a predicted ±4% accuracy for the sensor. Predicted performance curves are shown in Figure 38. The operational service life of the MSOGS is estimated at 8,000 flight hours.

The F-22 will use a non-dilution breathing regulator and G (BRAG) valve with positive pressure breathing for G capability (Figure 39). The breathing regulator will maintain a minimum of 1 in-Wg of mask cavity pressure. Above 39,000 feet the positive pressure schedule increases to 70 mm Hg (Figure 40). During high

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G maneuvers the BRAG valve will limit mask pressure to 60 mm Hg. Pressure relief provisions will be incorporated to limit mask pressure to 75 mm Hg. In the event of loss of OBOGS below 10,000 feet cabin altitude, the BYPASS mode will provide filtered air to the pilot. In the BYPASS mode the system will provide air at 40 LPM for pilot breathing.

The concentrator will operate at two speeds (a fast and a slow speed). Below a cabin altitude of 11,000 feet (changed from that shown in Figure 37) with AUTO selected the system switches between fast and slow speeds to maintain the average oxygen concentration below 60% to eliminate the occurrence of acceleration atelectasis. Above a cabin altitude of 11,000 feet the concentrator will operate at the fast cycle speed to maximize the oxygen concentration. Selecting MAX on the breathing regulator at any altitude will cause the concentrator to operate in the fast cycle speed only.
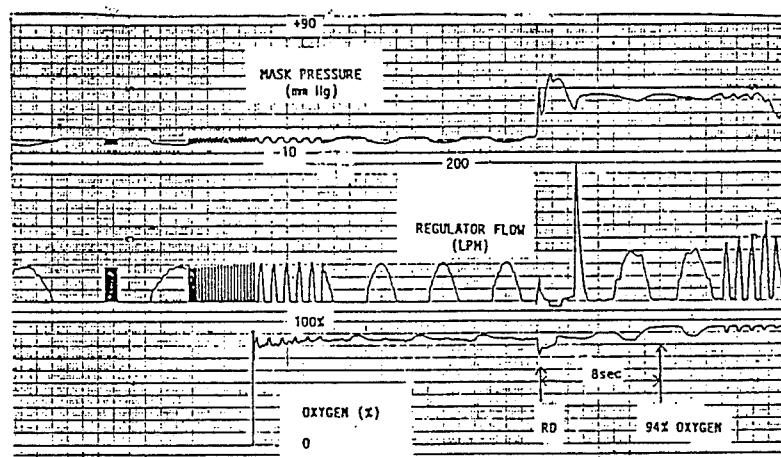
Figure 36. F-15E Rapid Decompression Data, 20,000ft to 80,000ft.

The system will not have a backup oxygen system. Instead, a seat-mounted, manually activated regulated emergency oxygen system (REOS) with a capacity of 100 NL will be available. An automatic warning signal will be issued to the pilot if the cabin altitude exceeds 25,000 feet. Upon receiving this signal the pilot will be required to manually activate the REOS. Once activated the REOS will flow oxygen until depleted. Also, the REOS will be activated automatically during the ejection sequence.
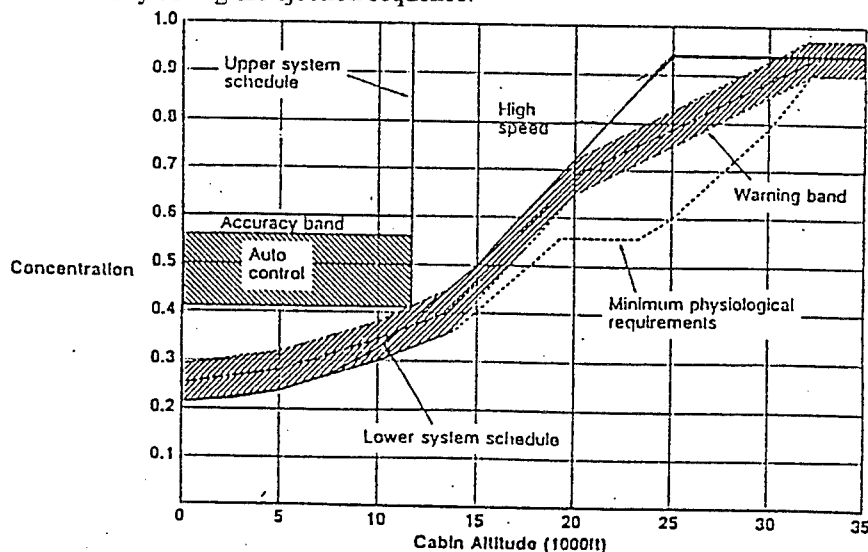
Figure 37. F-22 Oxygen Schedule.

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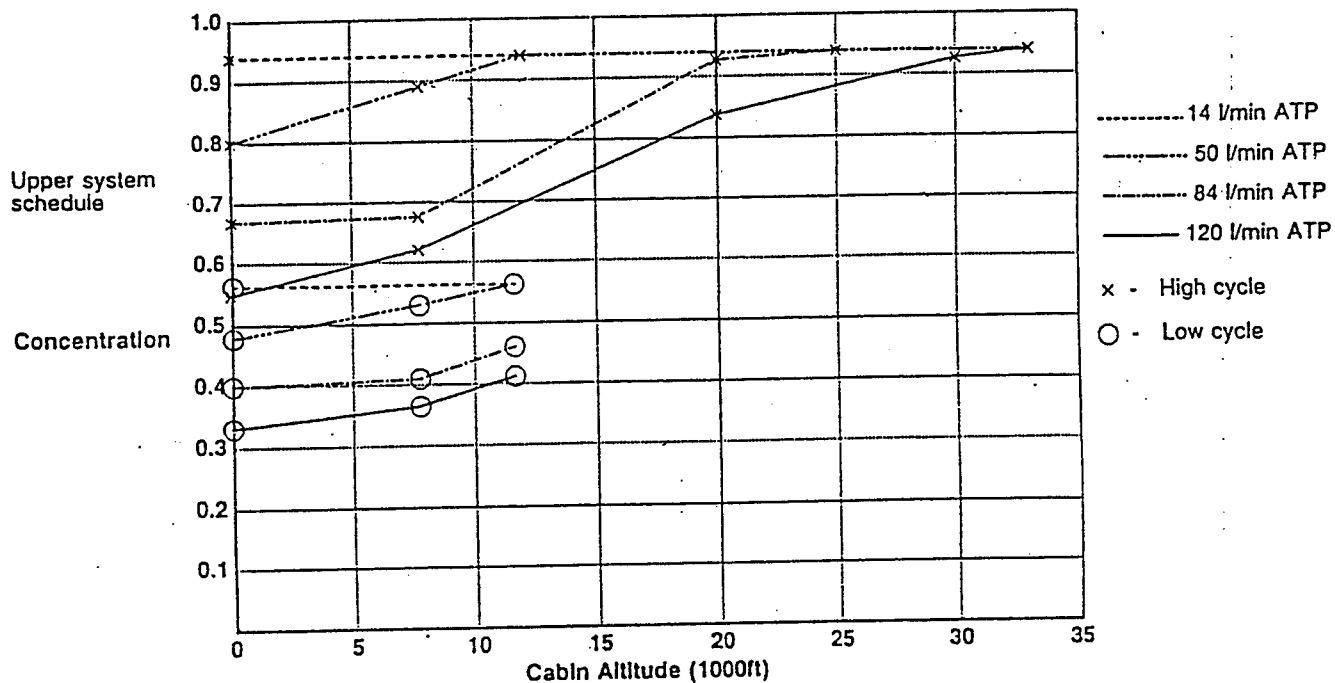
Figure 38. Predicted F-22 MSOGS Performance.
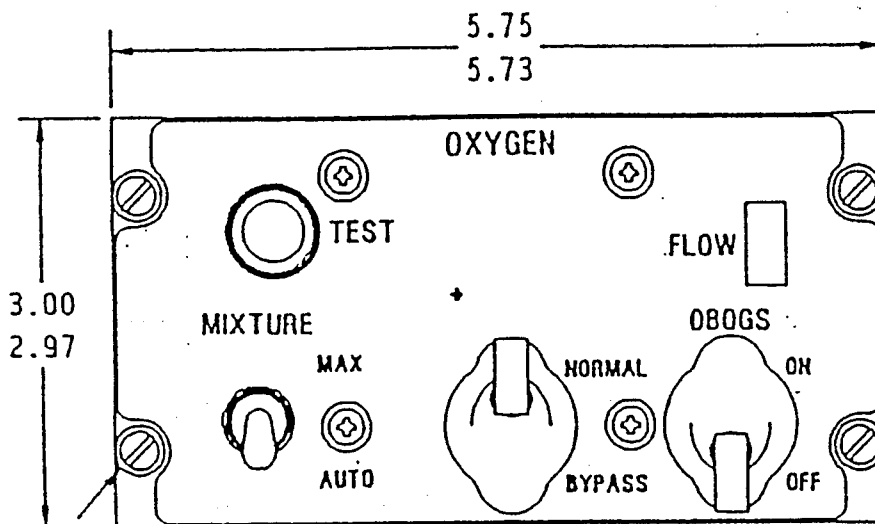
Figure 39. F-22 Breathing Regulator and Anti-G Valve.

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Future Oxygen Generation And Related Technologies

High Performance MSOGS (HP-MSOGS) A novel molecular sieve oxygen concentrator was developed which is capable of generating oxygen concentrations of up to 99.7% directly from compressed air.[15,16] This technology was patented by the Armstrong Laboratory, Brooks AFB, Texas in 1989.[17] The process is comprised of two zeolite molecular sieve beds and two carbon molecular sieve beds, and operates in the same manner as a standard molecular sieve oxygen concentrator (Figure 41). The highest oxygen purity from a standard molecular sieve oxygen concentrator is limited to 93-95% because the zeolite molecular sieve can not discriminate between oxygen and argon, hence both are concentrated. In the HP-MSOGS the zeolite molecular sieve removes nitrogen and the carbon molecular sieve removes argon, resulting in an oxygen purity of up to 99.7%. Presently, a 10 SLPM device has been fabricated and operated. This concept could be used on future fighter aircraft to generate aircrew breathing gas and fill to an integral backup oxygen system. Also, the US Air Force has awarded an exclusive license for this technology to a commercial firm. Commercial uses of this technology include hospital operating rooms, welding, metal cutting, and chemical processes requiring oxygen.

Ceramic Oxygen Generating System The basic mechanism for ceramic oxygen generation is the transport of oxygen ions across a zirconia ceramic membrane (Figure 42). The ceramic must be heated to a high temperature (≈1200°C). Oxygen molecules ionize on the air side of the plate, diffuse through the zirconia matrix, and recombine on the opposite side. An electrical current is required to transport the oxygen ions through the zirconia matrix. Potentially, the system could produce nearly pure oxygen (99.9%). The current disadvantages of the system are high power consumption and high operating temperatures. Presently, the US Air Force and Navy are involved in a joint program to further develop this technology.
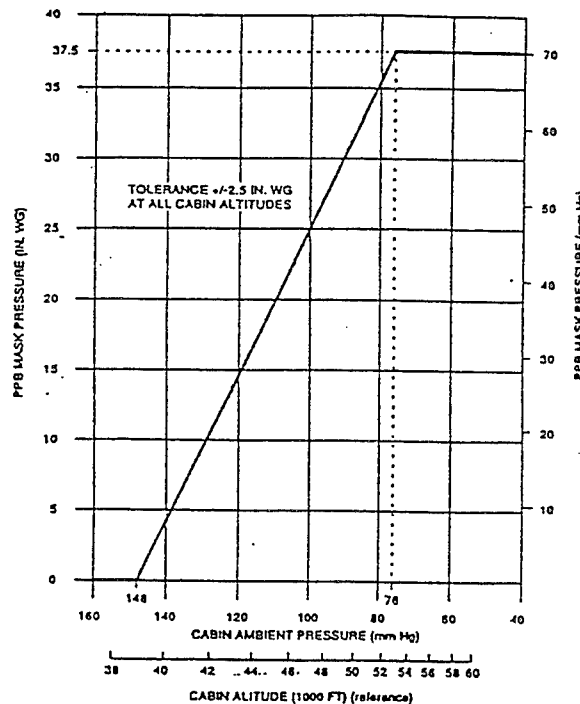
Figure 40. F-22 Pressure Breathing Schedule for Altitude.

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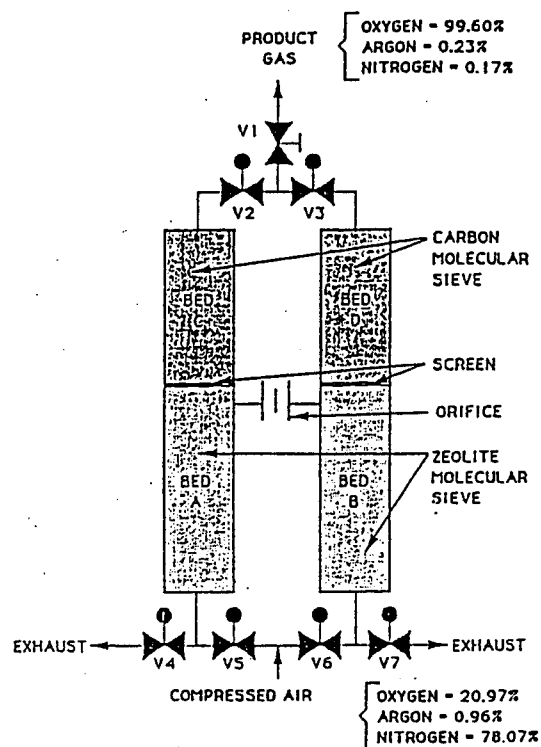
Figure 41. High Performance Molecular Sieve Oxygen Generating System.

Hybrid Oxygen System The hybrid oxygen system concept involves liquefying and storing oxygen generated by a MSOGS/OBOGS (Figure 43).[18] A laboratory demonstrator system has been constructed which uses helium coldheads to provide the cryogenic refrigeration. Compressed air cooled to cryogenic temperatures is passed through a heat exchanger to liquefy the oxygen produced by the MSOGS. The system was designed in an open loop configuration. Hence, after the compressed air is used to liquefy the oxygen it is exhausted to the atmosphere. Presently, MSOGS systems generate oxygen based on the demand placed on the system. With the hybrid oxygen system concept oxygen is continuously generated and that portion not consumed is stored. This concept could be used on multi-crew and aeromedical evacuation aircraft to fill and top off liquid oxygen converters. Laboratory testing of the hybrid oxygen system concept has been successful during operation at ground level. Work on the hybrid oxygen system concept was initiated at the Armstrong Laboratory, Brooks AFB, Texas in 1985.
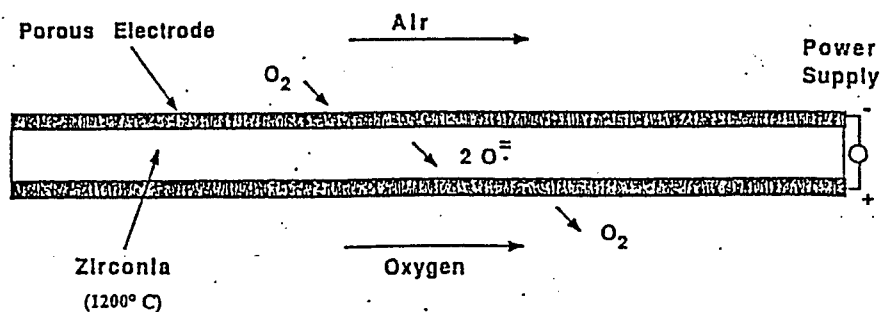
O_2 ionizes at the negative electrode

$O^=$ anions diffuse through the ceramic

O_2 molecules reform at the positive electrode

Figure 42. Simplified Schematic for a Ceramic Oxygen Generation Concept.

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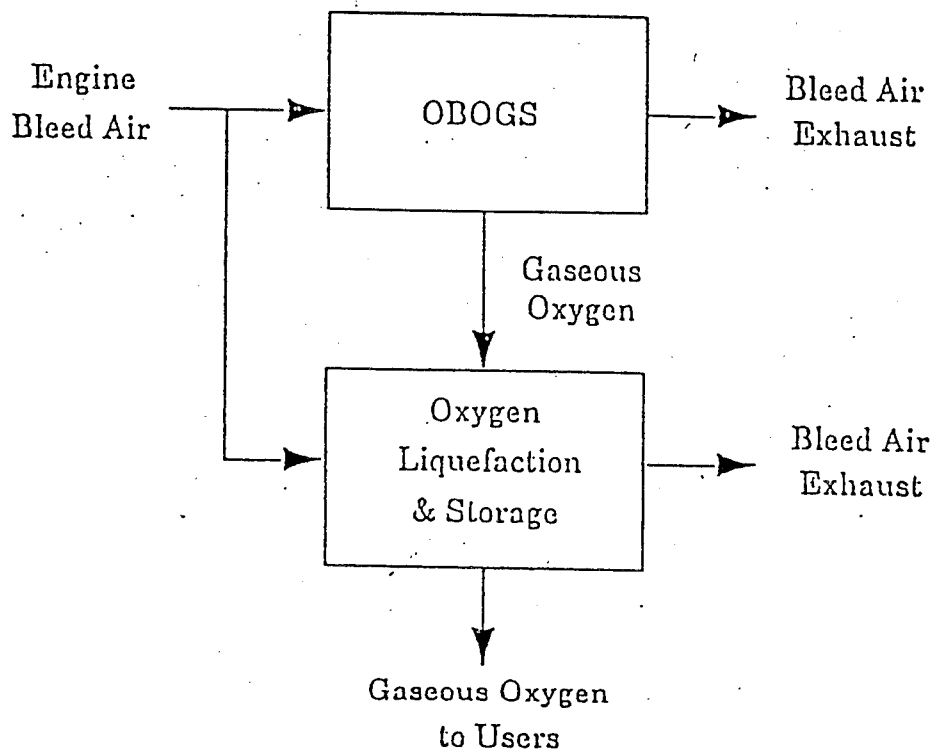
Figure 43. Simplified Schematic of the Hybrid Oxygen System Concept.

"Smart" MSOGS A smart MSOGS is controlled by a set of computer algorithms. The smart system automatically adjusts concentrator operating parameters to accurately control product oxygen concentration while minimizing bleed air consumption. One approach allows the computer algorithms to continuously adjust concentrator cycle time. For example, if the aircrew requires a lower oxygen concentration during certain phases of the mission (low altitude operations), the control software would slow the MSOGS cycle time. This concept could be used on future aircraft to precisely control the oxygen concentration and minimize consumption of engine bleed air. Reduced bleed air demand increases aircraft thrust and improves fuel economy. Laboratory testing of the smart MSOGS concept has shown that bleed air consumption can be reduced up to 60% while the oxygen concentration is precisely controlled. Work in this area was initiated at the Armstrong Laboratory, Brooks AFB, Texas in 1989.

Conclusions

MSOC technology has evolved into the dominant technology for generating oxygen on-board military aircraft. A comparison of aircraft molecular sieve oxygen generating systems is given in Table 1. Each new system is different from previous systems because MSOC technology must be integrated into the airframe, must be optimized for each aircraft (to minimize weight and space and maximize performance), and continues to evolve. Future MSOCs for fighter aircraft could incorporate high performance and smart MSOGS technologies. These systems would have a self-charging 99% oxygen backup capability and an on-board computer for precise control of oxygen concentrations and minimization of engine bleed air consumption. Future MSOCs for transport aircraft could potentially combine the hybrid oxygen system concept, and high performance and smart MSOGS technologies. The result would be a self-charging liquid oxygen system to support multi-man crews and patients. Ceramic oxygen generation technology may also be applied in the future.

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Table 1. Comparison of Current and Future Molecular Sieve Oxygen Generating Systems.							
			Aircraft System				
System Characteristic	AV-8B (1981)	F-16 Prototype (1982)	B-1B (1985)	TLLS (1986)	OGADS (1988)	F-15E (1990)	F-22 (1996)
Aircrew Size	1	1	4-6	1	2-3	2	1
Regulator Type	non-dilution	non-dilution	non-dilution	non-dilution	non-dilution	dilution	non-dilution
PPB for G	No	No	No	Yes	No	Yes	Yes
Ceiling (feet)	42,000	50,000	42,000	60,000	50,000	50,000	-
PBA (mm Hg)	30	30	19	70	30	30	70
Maximum Oxygen %	93-95	93-95	93-95	93-95	93-95	93-95	93-95
Composition Control	none	one speed with product venting	none	two speed	two speed	N/A	two speed
Oxygen Monitor	polarographic	polarographic	none	fluidic	fluidic	zirconium	zirconium
Molecular Sieve	5AMG	5AMG	OXYSIV-5	MG3	MG3	OXYSIV-5	OXYSIV-5
Design Flow Rate (ALPM)	13	50	160	60	120	100	60
System Weight (lbs)	41	~41	97	39.3	-	54	~40
BOS* Capacity (NL)	0	200	2800	190	850	260	0
BOS Oxygen Concentration	-	99.5%	99.5%	99.5%	99.5%	93%	-
EOS+ Capacity (NL)	(REOS) 200	50	50	100	50	50	(REOS) 100
EOS Oxygen Concentration	93 or 99.5%	99.5%	99.5%	99.5%	99.5%	99.5%	99.5%

* Backup Oxygen System

+ Emergency Oxygen System

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Discussion

MAJ. CAULKINS: It's interesting that when you look at all the MSOGS systems that you showed for different aircraft, the F-22 is only one of two aircraft that doesn't have a backup system. Will you comment on that?

MAJ. MILLER: At meetings we have noted our preference for a backup system. The backup system would provide oxygen if there were upsets in the MSOC system with regard to pressure or temperature, so that your sieve oxygen concentration falls. If the backup system comes on-line, like in the F-15, it will reduce the need to use the emergency oxygen to solve the problem. Now, if the F-22 pilot were to get a low oxygen light, he's got to activate the emergency oxygen and that may cause the mission to be aborted because he will use up his emergency oxygen supply over time. I think the additional weight was the reason the backup system was not selected for the F-22?

MS. MCGARVEY: The weight of a backup oxygen system was about 15 pounds. The primary purpose for having the backup oxygen is because of the ECS transient temperature and/or pressure effect on the OBOGS. Because this is a new aircraft system, we found that you could control the temperatures and the pressures out of the ECS so as to guarantee OBOGS function. In addition, we looked at what the Navy was doing. The Navy doesn't have a lot of backup oxygen systems. The F-18 doesn't, the AV-8 doesn't. So if you look across the Department of Defense, there isn't a lot of rationale for adding a backup system.

MAJ. CAULKINS: They're different concentrators.

MS. MCGARVEY: They're still MSOGS concentrators; you're still looking at those kinds of technology. Also, when we explored the issue about decompression, we found that there are very few rapid decompressions in fighter aircraft which would cause you to instantaneously need emergency oxygen. We decided to address the causes of the loss of cabin pressure by providing better sealing capabilities and providing additional pressurization warnings for the pilot. We also have a cabin pressure check on the ground to make sure that you're actually getting a seal.

When we reviewed the requirements for flight above 50,000 feet, there were some very good reasons to have 100% oxygen or something in excess of the 93% oxygen to really protect the person. We looked at the system in the F-15E where you have a plenum that contains 93% oxygen and that didn't meet our needs, especially since our contractor indicated that, as a result of the inaccuracy in the zirconium sensor, we couldn't even guarantee ourselves a 93% oxygen concentration. In addition, the TLSS system had a standby backup high-pressure system of 100% oxygen which leaked badly. So, for all those reasons we decided to increase the size of the 100% emergency oxygen cylinder and use a demand regulator system.

MAJ. MILLER: The crews didn't like the F-15E system when it first came out because there were some software bugs. The built in test features and computer control on the concentrator initially resulted in indications of system problems in flight. The contractor has finally worked out the bugs and the aircrew now really like the system. I have been told that the backup oxygen system (BOS) does activate quite frequently during flights. The 93% BOS comes on-line when there are interrupts in pressure and then it drops out and goes back to MSOGS following the temporary problem. So, the BOS on the F-15E is regularly used and the crews like the system.

MAJ. CAULKINS: Are there any comments on the Eurofighter 2000 system?

PROF. ERNSTING: I'd just like to make a couple of comments. The UK version of the AV-8B used the same technique as the group here did for dumping excess product gas and is close loop controlled with a fluidic sensor. We have had the system operating in 100 airplanes for the last four or five years. Its reliability is out of this world compared with what we used to have with LOX in the Harrier force. It's well liked by the aircrew.

We could also discuss the use of terms "backup" and "emergency". I don't think it's very fair to say the United States Navy doesn't have a backup system in the AV-8B. The system contains 200 liters that can be used; the problem is that you can't reverse it, once activated. Whereas in the RAF version of the AV-8B, the Harrier GR5, we have reversible control so the pilot can select emergency backup supply. He has an indication of the quantity and, at any time, can

switch back to the concentrator gas if the system is working adequately. The system was developed in the 1980s. One last point I'd make is that the final version of the Eurofighter will have a highly sophisticated computer control of its MSOG product gas.

DR. BOMAR: Can you comment on the system that the Air National Guard is now putting on the F-16, which is not the same one as was originally put on the F-16A. There's a version flying on the Air National Guard F-16C now. Is that not correct?

MAJ. MILLER: I would call it a prototype system flying on the Air National Guard aircraft. It's called a slimline 5 system, made by Litton, and they're in flight trials now. I think the jury is still out on that system. It has an emergency oxygen system, but not a backup system.

DR. BOMAR: But it has two plenums though.

MAJ. MILLER: Right, there are two plenums in the piping that runs between the concentrator and the cockpit to provide a little extra gas to the crew member if he should lose OBOGS. However, the supply doesn't last very long and they're looking at replacing it with something else. So, I have a feeling that before they go to production it could get modified. I believe the slimline 5 is flying on the Navy F-18 aircraft. What they'd like to do is put this low cost system on the F-16. There were some problems noted during the qualification testing we conducted; more specifically, it looked good as a one man system, but not too good as a two man system and they want the system to work on both one seat and two seat aircraft. They're now completing the flight trails. So, I think we'll just have to wait until the flight testing is over before we can comment further on that system.

Cabin Pressurisation Schedules - Acceptable Compromises

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Introduction

The concept of pressurising the cabin of an aircraft so that the crew and passengers are not exposed to low environmental pressure at altitude is nearly as old as powered flight. It was first considered during World War I and by 1921 the US Army Air Corps had conducted a test flight in which the pilot of the aircraft was enclosed in a tank which was pressurised with air. The flow capacity of the discharge valve fitted to the tank was, however, totally inadequate so that during the flight at 3,000 feet, the pressure within the tank increased to the equivalent of 7,000 feet below sea level. The pilot suffered severe otitic barotrauma and the temperature within the cabin rose to 66°C. This failure of engineering design delayed further attempts in the United States at cabin pressurisation. Aircraft fitted with experimental pressure cabins were, however, developed and flown during the early 1930s by several European nations, including Germany and France. The parallel concept of the sealed cabin pressurised with oxygen carried with the vehicle was, however, developed and exploited by high-altitude balloonists such as the Belgian Piccard who ascended to an altitude of 54,120 feet in 1932, and Settle and Fordney in the United States who ascended to an altitude of 61,237 feet in 1933.

The early attempts to pressurise the crew compartment of an aircraft revealed the major factors which had to be taken into account in producing an acceptable pressure environment for the occupants. It was recognised early in the development of cabin pressurisation that the pressure to be maintained within the cabin during flight was a function of the physiological effects of altitude, specifically hypoxia, and whether the occupants would be using supplemental oxygen. The earliest studies had shown the importance of adequate control of the differential pressure of the cabin. It was also recognised that the strength of the pressure-holding structure was a vital consideration, both with regard to the integrity of, and the increased weight penalty imposed by, the structure. The possibility of a sudden failure of the pressure cabin in flight was considered and led to the extensive studies of rapid decompression performed in the late 1930s and the 1940s. The importance of adequate control of the ventilation of the pressure cabin and the temperature within it were established. The fundamental aeromedical requirements for the pressure cabin were specified in the classic report by Armstrong in 1935 (2). These requirements were embodied in the design of the XC-35 sub-stratosphere airplane which was built by Lockheed for the US Army Air Corps. This aircraft completed a very successful flight test programme in 1937 which provided a firm basis for the pressurisation of the crew and passenger compartments of future aircraft.

World War II saw the development and introduction into service in the United States and the United Kingdom of fighter and bomber aircraft equipped with pressure cabins. The requirement for minimum aircraft mass and the likelihood of rapid decompression of the cabin due to enemy action led to the adoption of a low pressure differential for fighter aircraft - typically of the order of 2.0 to 2.75 Lb in^{-2}, whilst the advantages of being unencumbered with oxygen equipment throughout flight led to the selection of a pressure differential of 6.5 to 7.5 Lb in^{-2} for bomber aircraft. The aeromedical requirements which determined the pressurisation schedules of the military aircraft constructed during World War II were well summarised by Lovelace and Gagge (12). These set the maximum cabin altitude without supplemental oxygen at 10,000 feet [5,000 feet for night vision] and the maximum allowable cabin altitude to avoid decompression sickness (aero-embolism) at 30,000 feet. The use of a limit to the Relative Gas Expansion (RGE) as the safety criterion for a sudden decompression of the cabin was widespread. Thus Lovelace and Gagge advocated a maximum RGE of 2.3 for a fighter cockpit with a volume of 50 cubic feet (12).

The late 1940s saw the adoption of cabin pressurisation for all high-altitude aircraft. The maximum cabin altitude allowed in the high-differential pressure cabins of bomber and commercial aircraft in which cabin air was

breathed throughout flight was set at 8,000 feet. In fighter aircraft the weight and hence aircraft performance penalties of a high-differential pressure cabin such that the crew could breathe air throughout flight were unacceptable. Furthermore it was considered that the threat to the integrity of the pressure cabin by enemy action and the possible ensuing decompression should be taken into account in this type of aircraft. Thus, it was generally accepted that the crew of fighter aircraft would wear oxygen equipment throughout flight so that the magnitude of the cabin pressure differential in this type of aircraft could be considerably less than if the maximum cabin altitude was limited to the 5,000 to 8,000 feet required when breathing air. The maximum cabin altitude for this type of aircraft was set at 25,000 feet (McFarland (14)). There was a divergence in the pressurisation schedules subsequently developed for fighter aircraft between the United States and the United Kingdom (Figure 1). The concept of the isobaric pressurisation schedule in which with ascent of the aircraft the cabin altitude is held constant at the value at which pressurisation commences until the maximum cabin differential pressure is attained was adopted in the United States. The United Kingdom, in contrast, employed in all its indigenous fighter aircraft a pressurisation schedule in which the cabin altitude increases progressively with ascent of the aircraft with the maximum cabin pressure differential not being attained until the aircraft is at an altitude of 35,000 - 40,000 feet [the cabin differential pressure with an isobaric pressure schedule is constant at all aircraft altitudes above about 23,000 feet].
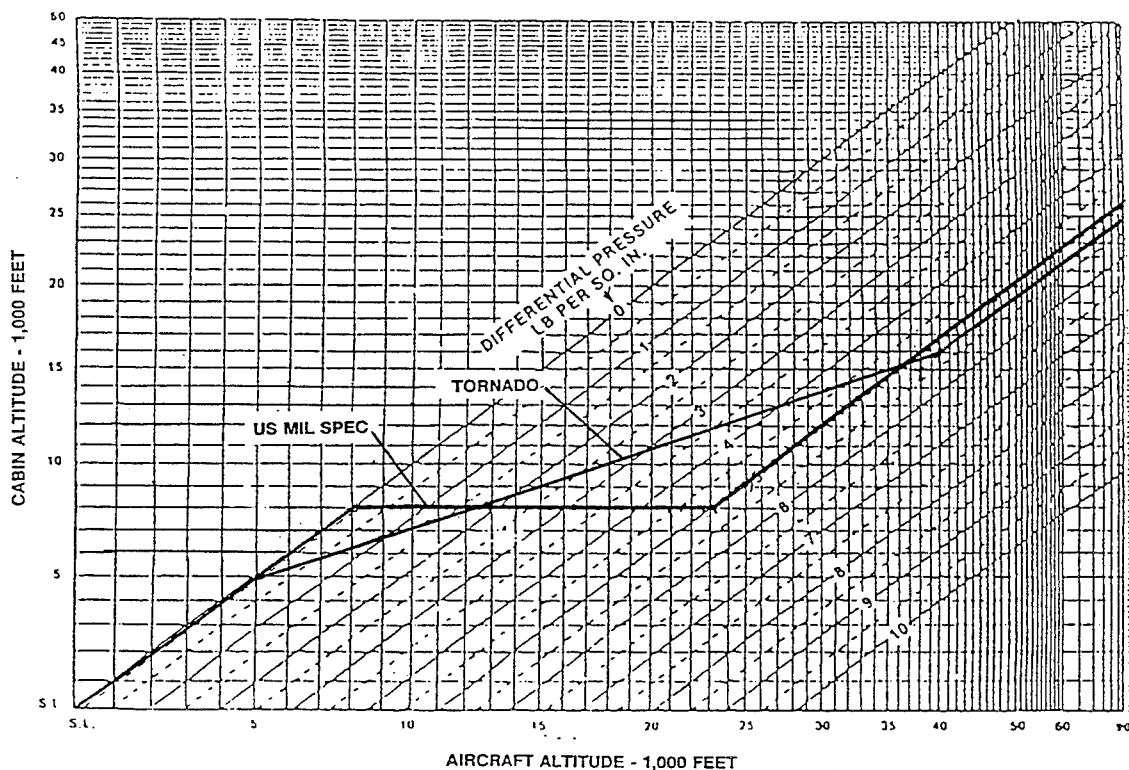
Figure 1. Divergence in pressurisation schedules for fighter aircraft, United States and the United Kingdom.

The pressurisation schedules employed in modern fighter aircraft represent a series of compromises between various conflicting aeromedical, engineering and operational requirements which are examined in this paper. These aeromedical requirements relate to hypoxia, decompression sickness, otitic and sinus barotrauma and the effects of failure of the pressure cabin. The engineering requirements are principally the need to minimise the mass of the aircraft, to maximise the reliability and life of the cabin and to reduce the risk of losing the aircraft and its crew in the event of a failure of the pressure cabin due to enemy action.

Aeromedical Requirements for the Pressurisation of Combat Aircraft

Avoidance of Hypoxia

It is possible to maintain the alveolar oxygen tension at the value associated with breathing air at sea level by progressively enriching the inspired air with oxygen at altitudes up to 33,000 feet. However, the rate at which the function of the central nervous system is impaired by an interruption of the oxygen supply, so that the aircrew member reverts to breathing air increases progressively as the altitude is raised above 15,000 feet. The time available at an altitude of 20,000 feet for an individual to recognise that his oxygen supply has ceased and for him to carry out the appropriate corrective action is approximately three times greater than the time available at an altitude of 25,000 feet. Furthermore, the reduction of the inspired oxygen tension produced by a given fractional inboard leak of air due to an ill fitting oronasal mask increases with increase of altitude. Thus, although it is theoretically possible to maintain a normal sea level alveolar oxygen tension at altitudes of up to 33,000 feet by increasing the concentration of oxygen in the inspired gas, these considerations suggest that both the incidence of hypoxia and its severity will increase with increase of altitude above 15,000 feet. The incidence of hypoxia accidents in unpressurised aircraft in World War II was 6 times greater for flights at 25,000 feet as for those at 20,000 feet, whilst, when the altitude of flight was 30,000 feet, the incidence was nearly 70 times that at 20,000 feet. The experience of the Royal Air Force over the last 20 years has amply confirmed these war-time observations, with the incidence and severity of hypoxia accidents rising significantly with increase of cabin altitude between 20,000 feet and 25,000 feet. It is concluded that in order to minimise the risks of hypoxia arising from an ill fitting mask or a malfunction of the oxygen delivery system, and to provide adequate time for the recognition and correction of a malfunction of the oxygen system, the cabin altitude should not exceed 20,000 feet.

Avoidance of Decompression Sickness

Pressurisation of the cabin of a combat aircraft plays an essential role in preventing the occurrence of decompression sickness when the aircraft is at high altitude especially when the length of time at altitude extends to several hours. The experience of the incidence of decompression sickness during World War II led to the decision that the maximum cabin altitude in fighter aircraft should not exceed 25,000 feet (14). Subsequent experience in several air forces, including the United States Air Force (11) and the Royal Air Force (6), showed that there are occurrences of decompression sickness at cabin altitudes below 22,000 feet and that a few cases, occasionally severe, have occurred at cabin altitudes as low as 18,000 feet. A review of the cabin pressurisation schedules for combat aircraft conducted in the UK in the late 1960s concluded that whilst the maximum safe altitude for the avoidance of decompression sickness was 18,000 feet, very few cases of serious decompression sickness are likely to occur below a cabin altitude of 22,000 feet. With the general adoption of a cabin differential pressure of 5.0 Lb in^{-2} in USAF aircraft since the early 1960s and in the RAF since the late 1960s [which provides a cabin altitude of 18,500 feet at an aircraft altitude of 45,000 feet], and the relatively rare occasions on which fighter aircraft operated at altitudes above 45,000 feet for any significant length of time, the incidence of decompression sickness in fighter aircraft became negligible [the incidence remained high however in low cabin differential pressure reconnaissance aircraft in which the crew are exposed for several hours to cabin altitudes as high as 26,000 to 28,000 feet].

The extensive studies conducted by the Armstrong Laboratory using Doppler ultrasound to detect and semi-quantify the occurrence of venous gas emboli in the right side of the heart have demonstrated that significant quantities of venous gas emboli occur in subjects exposed for several hours to altitudes as low as 15,000 feet when the inspired gas contains nitrogen (19). This group of investigators have advocated that the cabin altitudes of combat aircraft which may fly at very high altitudes for several hours should not exceed 16,000 feet (19). Although serious symptoms of decompression sickness are extremely rare at cabin altitudes below 20,000 feet, serious

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decompression sickness could well develop rapidly after the rapid decompression to high altitude of a pilot with severe venous gas emboli. Further experimental evidence is urgently required with respect to the possibility and the incidence of symptoms of decompression sickness at altitudes between 16,000 and 20,000 feet under the conditions to be expected in modern and future combat aircraft, viz. the crew performing light work and breathing gas containing 30-40% nitrogen (4). At present it is considered that decompression sickness should be avoided by not allowing the cabin altitude to exceed 18,000 feet. Short duration exposures to cabin altitudes as high as 20,000 feet are very unlikely to produce a significant incidence of decompression sickness.

Avoidance of Gastro-intestinal Disturbances

Abdominal discomfort or pain due to the expansion of gas within the gastro-intestinal tract is extremely rare in aircrew at altitudes below 25,000 feet. A small proportion of experienced subjects will develop significant abdominal discomfort on exposure to altitudes above 30,000 feet. A maximum cabin altitude of 25,000 feet will prevent the occurrence of abdominal discomfort due to expanding gas in the stomach or intestines.

Ventilation of the Middle Ears and Sinuses

Gas must flow into the middle ear and the paranasal sinuses from the nasopharynx and the nose during descent if the pressures in these cavities are not to fall below that of the environment. In healthy subjects, gas always flows freely into the sinuses but the majority of aircrew have to perform a purposeful act in order to introduce gas into the middle ear cavity. Swallowing and movement of the lower jaw produces effective ventilation of the middle ear in some subjects, but at least 50% of experienced aircrew have to occlude the external nares and raise the pressure in the nasopharynx to open the Eustachian tube so that gas can flow into the middle ear. The majority of these individuals use the Frenzel manoeuvre, a few use Valsalva's manoeuvre, although the latter is an undesirable technique for aircrew. Failure to ventilate the middle ear during descent gives rise to increasing discomfort in the ear and deafness, and eventually to otitic barotrauma. The discomfort produced in the ear by descent and the need to occlude the nostrils to perform the Frenzel manoeuvre can distract the aircrew member from his primary task. It is vital to avoid otitic or sinus barotrauma, which often prevents the individual flying for several days. It is highly desirable that in war aircrew with an upper respiratory tract infection can continue to fly operationally. All these considerations argue for the minimum increase and decrease of cabin altitude during flight. Other factors which have been discussed in the introduction to this paper do not allow this solution in high-performance combat aircraft, although the isobaric cabin pressurisation schedule of the United States Military Specification for low pressure differential cabins (17) provides the ideal of no change of cabin altitude during aircraft manoeuvres between altitudes of 8,000 and 23,000 feet (Figure 1). The rate of increase of pressure within the cabin on descent of a combat aircraft should be as low as possible to avoid ear discomfort and to minimise the frequency with which the nose must be occluded to perform the Frenzel manoeuvre and the incidence of otitic barotrauma. Aeromedical advice in the United Kingdom has recommended (15) that on descent, the rate of increase of absolute pressure in the cabin should not exceed 2.0 Lb in^{-2}/min over a change of pressure of 1.0 Lb in^{-2}. This advice was based on the incidence of otitic barotrauma in flight and hypobaric chamber studies.

Integrated Aeromedical Requirements

It is considered that in respect of the avoidance of hypoxia and decompression sickness, the cabin altitude of a combat aircraft should not exceed 20,000 feet when the aircraft is at its service ceiling. Furthermore, it is highly desirable that the cabin altitude does not exceed 18,000 feet on the vast majority of operational sorties. Avoidance of ear discomfort and the minimisation of the frequency with which the nostrils have to be occluded to introduce gas into the middle ear, and of the risk of otitic barotrauma, requires that the rate of increase of cabin pressure during descent of the aircraft should be as low as possible, and ideally should not exceed 2.0 Lb in^{-2}/min for an increase of pressure of 1.0 Lb in^{-2}.

Aeromedical Aspects of Rapid Decompression of the Cabin

The pressurisation of the cabin of a combat aircraft may fail due to cessation of the flow of air into the cabin (due to engine failure or malfunction of the environmental control system), opening of the cabin outlet valve

(due to a failure of the control system or selection by the pilot) or a defect in the pressure cabin structure (due to enemy action, human error, mechanical failure or as a prelude to ejection). The cabin altitude-time profile on decompression is determined by the ratio of the effective area of the defect in the structure to the volume of the cabin, the magnitude of the air flow into the cabin, the magnitude of the aerodynamic suction and the altitude-time profile of the aircraft, including the initial aircraft altitude, the time taken to initiate descent and the rate of descent of the aircraft.

The hazards of a rapid decompression of the cabin include physical injury due to the structural failure and very high air flow velocities in the cockpit, lung barotrauma, hypoxia, decompression sickness, cold injury and hypothermia. Of particular concern in relation to the cabin differential pressure at the instant at which the decompression occurs are pulmonary barotrauma and hypoxia.

Pulmonary Barotrauma

Free venting of the gas in the lungs on a sudden reduction of the environmental pressure is hindered by the resistance to the flow of gas from the alveoli through the airways to the mouth and nose and by the resistance imposed by the breathing equipment (5, 13). If the rate and range of the decompression are large, then the expanding lung gas will be unable to escape and the consequent increase in the pressure difference between the alveolar gas and the surface of the chest and abdomen (the trans-thoracic pressure) will expand the lungs and chest wall and force descent of the diaphragm. Excessive distension of the lungs will tear the lung parenchyma and gas will enter the pulmonary tissues and, more seriously, enter torn pulmonary vessels so that gas passes into the left side of the heart and hence into the systemic circulation (18). Surgical emphysema in the mediastinum and neck and pneumothorax can also occur. A trans-thoracic pressure difference of the order of 80-100 mm Hg is required with the respiratory muscles relaxed to distend the lungs to the extent that the lung parenchyma is torn (10).

Rapid Decompression with the Glottis Closed

The worst case with respect to lung damage on a rapid decompression is when the glottis is closed so that gas cannot escape from the respiratory tract. The transthoracic pressure created by a decompression with a closed glottis is determined by the degree of inflation of the lungs at the instant of decompression and the ratio of the initial to final cabin pressures. Thus a decompression from 16,000 feet to 38,000 feet with the lungs at functional residual capacity and the glottis closed will produce a transthoracic pressure of 50 mm Hg (5, 13). The chance of the glottis being closed at the instant that a rapid decompression occurs is usually considered to be so remote that this risk is not considered in determining cabin pressurisation schedules.

Lung Damage with Free Venting of Gas from the Respiratory Tract

Experiments using a variety of animal models (18) and man (5, 9, 13, 16) have provided information on the conditions of rapid decompression (pressure range and rate of decompression) which are safe and those which may give rise to lung damage when the expanding lung gas can flow freely from the mouth and nose. A valuable approach to describing these conditions, which was developed initially by Violette (18), relates the probability of lung damage on rapid decompression to the ratio of the initial to the final pressures in the cabin and the time characteristic of the decompression as described by the ratio of the volume of the cabin to the area of the orifice though which it is decompressed. Violette defined, using a variety of animals, a curve (Figure 2) which separated decompressions which caused lung damage from those which did not. There is a paucity of experimental data with respect to rapid decompressions which can cause lung damage in man. The conditions of rapid decompression to which human subjects, wearing either no breathing equipment or equipment which allowed free venting of gas from the respiratory tract, have been exposed without any evidence of injury to the lungs are indicated in Figure 2 (5). No studies have been conducted in man under the conditions found by Violette to cause lung damage in animals. The relationship of the time of decompression to the variables employed by Violette (18) (Figure 2) can be calculated using the equations developed by Haber and Clamann (8) as shown in Figure 3. It may be seen from the latter that the decompression of the cabin of an aircraft with a cabin pressure differential of 5.0 Lb in^{-2} flying at 40,000 feet in 0.05 sec is very unlikely to produce lung damage provided that gas can escape freely from the
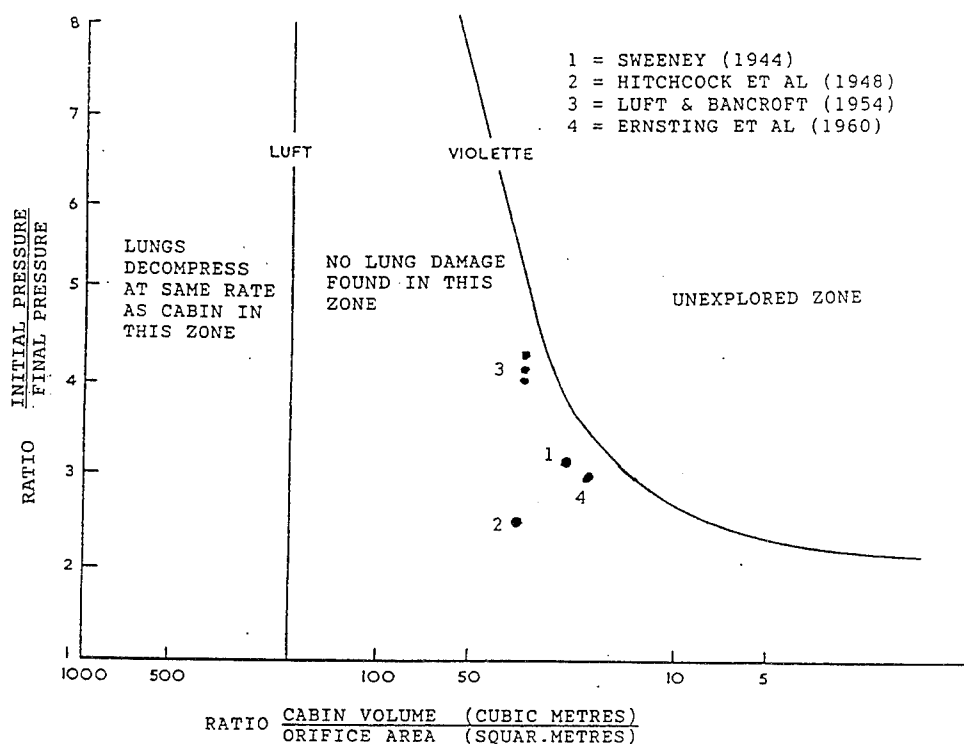
Figure 2. Decompression conditions and lung damage.
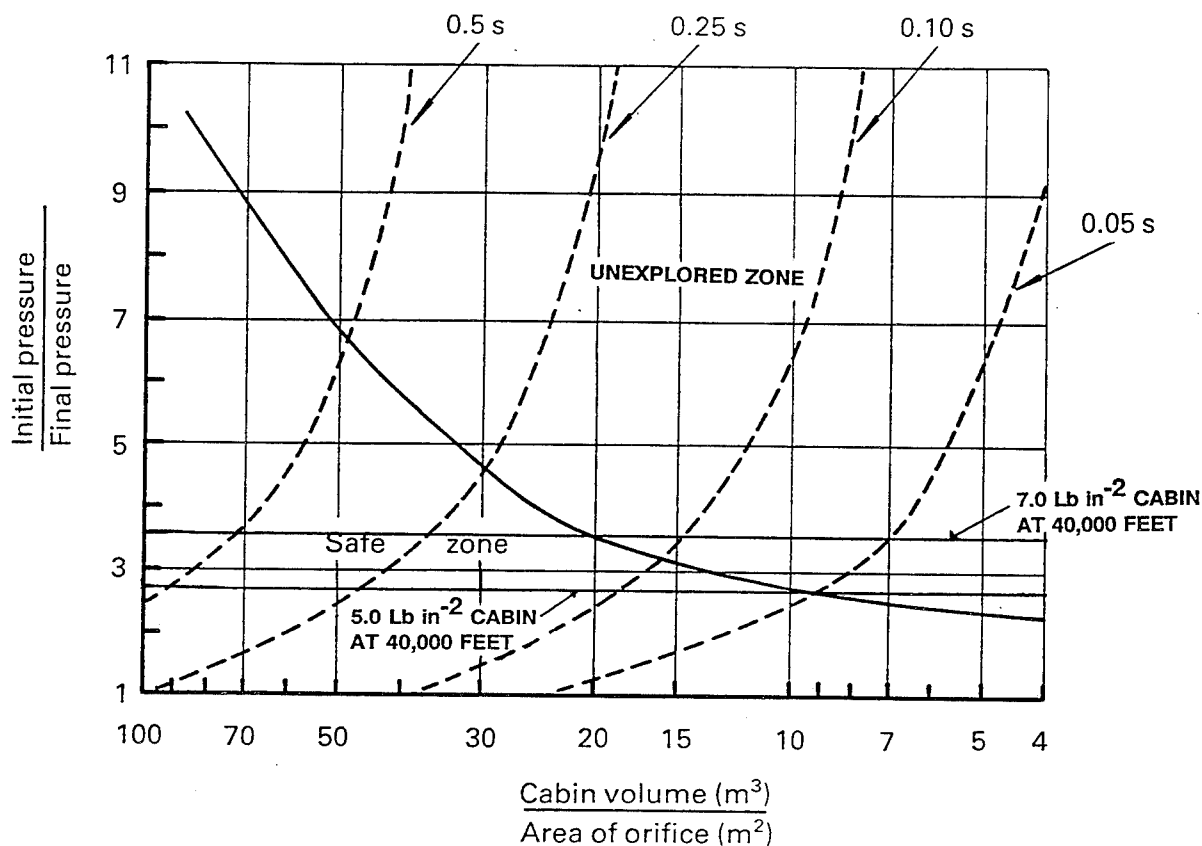
Figure 3. The relationship of time of decompression to variables employed by Violette (Figure 2) calculated using the equations developed by Haber and Clamann.

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respiratory tract. Even increasing the pressure range of the decompression from 5.0 to 7.0 Lb in^{-2} probably only produces a small increase in the risk of lung damage at a decompression time of 0.05 second and virtually no risk at a decompression time of 0.1 sec. Thus avoidance of the possibility of lung damage on a very rapid decompression such as occurs with the sudden loss of the canopy of the cockpit when there is no hindrance to the flow of gas from the respiratory tract requires that the differential pressure of the cabin should not exceed 5.0 Lb in^{-2}. Taking into account the uncertainties of the conditions of safe decompression as presented in Figures 2 and 3 and mindful that human subjects have not been exposed under controlled conditions to decompressions in the "zone of uncertainty", it is believed that the increased risk associated with a cabin pressure differential of 6.0 - 7.0 Lb in^{-2} is probably very low.

Potential for Lung Damage When Wearing Breathing Equipment

The conventional pressure demand valve system fitted to masks which comprises an inlet non-return valve and an outlet valve which is compensated to the pressure in the mask hose will impose a high resistance to the flow of gas from the lungs on decompression, and will increase the likelihood of lung damage on rapid decompression. Indeed, when used in a conventional pressure demand system, the compensated outlet valve of the mask will be held firmly shut throughout the decompression by the pressure exerted by the gas trapped in the delivery hose between the demand regulator and the inlet valve of the mask. Rapid decompression of the cabin of a combat aircraft when wearing a pressure mask such as the USAF MBU-5/P and the USAF/USN MBU-12/P is only safe because these masks do not seal well at high pressures. A leak from the mask during the decompression allows the pressure in the mask delivery hose to fall and the outlet valve of the mask to open. The increased hazard which arises when a well sealing mask such as the RAF series P/Q/V masks is worn necessitates the introduction of a method whereby the gas trapped between the regulator and inlet valve of the mask is free to expand without a significant increase in mask tube pressure, or the compensation of the outlet valve of the mask is controlled from the demand regulator so that the valve will open during a rapid decompression. Examples of the former type of solution are the presence of a bladder (typically one used to apply counterpressure to part or all of the chest and abdomen) connected to the delivery hose between the demand regulator and the inlet valve of the mask, or the provision of a compensated dump valve in the regulator. Many US and UK partial-pressure suit systems include a chest or trunk counterpressure bladder and all modern UK and most recent US pressure demand regulators are fitted with a compensated dump valve facility. The solution whereby the demand regulator provides the pressure signal which controls the opening of the outlet valve of the mask is widely employed in the UK man-mounted regulator systems and in French seat-mounted oxygen regulators. The current ASCC standard (1) for the behaviour of the breathing equipment on rapid decompression requires that the mask pressure (measured relative to environmental pressure) during a decompression with a decompression time of 0.1 sec does not exceed 41 mm Hg. This standard is probably conservative as short duration peak mask pressures as high as 60-80 mm Hg have occurred on the experimental rapid decompression of subjects wearing pressure demand equipment without harm. Research is required to understand better the behaviour of the lungs on very rapid decompression and to update the specification of acceptable mask cavity pressures during very rapid decompressions, both over the present cabin differential pressure of 5.0 Lb in^{-2} and at higher differential pressures up to at least 7.0 Lb in^{-2}.

The introduction of pressure breathing as a means of enhancing tolerance of high sustained accelerations (PBG) has brought with it the possibility that a rapid decompression could occur when the pressure in the respiratory tract is already raised to the order of 50-60 mm Hg. All the PBG systems at present being introduced into operational use employ a chest counterpressure garment to reduce or prevent lung distension during pressure breathing. The bladder of the chest counterpressure garment is connected into the delivery hose between the pressure demand regulator providing PBG and the oronasal mask. Rapid decompression when pressure breathing wearing this equipment produces very high pressures in the mask, respiratory tract and the bladder of the chest garment unless an additional dump valve facility is fitted. The bladder of the chest counterpressure garment of present PBG systems is fitted with a compensated dump valve which allows gas in the garment to escape on a rapid decompression and limits the amount by which the pressure in the bladder of the garment exceed the pressure in the mask (7). A moderate degree of overpressurisation of the chest counterpressure garment relative to the mask and the lungs is almost certainly desirable as it will tend to prevent excessive distension of the lungs during a rapid decompression. There is no experimental information available on the effects of rapid decompression on the lungs when exposed to +G$_z$ acceleration, either with or without PBG. This is a topic where further research is urgently

required to understand the behaviour of the lungs in these circumstances and to define acceptable pressures in the mask and chest counterpressure garment and the acceptable pressure difference between them.

Hypoxia on Rapid Decompression

The production and prevention of hypoxia on rapid decompression of the pressure cabin of a combat aircraft are fully described in the review by Ernsting (4) presented at this symposium. One aspect of this topic which is of direct interest in the present context is the effect which the magnitude of pressure change produced by a rapid decompression has upon the concentration of oxygen which must be breathed prior to the decompression in order to prevent the alveolar oxygen tension falling below 30 mm Hg at the end of the decompression. This requirement is described for a 5 Lb in^{-2} pressure cabin in Figure 1 of the paper by Ernsting (4) and is reproduced in Figure 4 of this paper. Increasing the cabin pressure differential to 7.0 Lb in^{-2} raises the concentration of oxygen which must be breathed prior to the decompression by 5-15% oxygen concentration depending upon the cabin altitude and the pressure breathing system employed to prevent hypoxia at cabin altitudes above 40,000 feet (Figure 4). Such an increase in the minimum concentration of oxygen markedly reduces the width of the limits of allowable concentration of oxygen to be provided by the breathing system at cabin altitudes above about 10,000 feet.
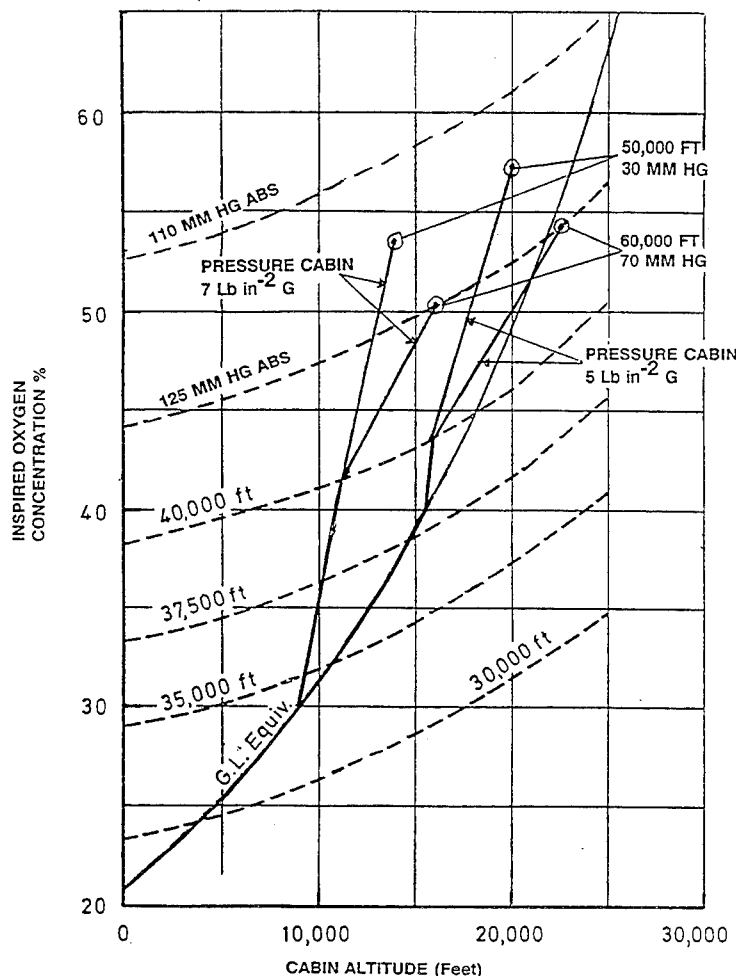
Figure 4. Effect that magnitude of pressure change produced by a rapid decompression has upon concentration of oxygen that must be breathed prior to decompression to prevent the alveolar oxygen tension falling below 30 mm Hg at the end of the decompression.

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Cabin Pressurisation Schedules

Mechanical Aspects

The pressure within the cabin of a combat aircraft is controlled by regulating the degree of opening of the discharge valve(s). A large fixed mass of conditioned air is delivered into the cabin by the aircraft Environmental Control System (ECS). The flow, temperature and humidity of this air and its distribution within the cabin are determined by the requirement to produce an acceptable thermal environment both for the crew and for the aircraft systems, especially the avionics equipment. To meet this thermal requirement, the mass flow of air in a typical high-performance combat aircraft is of the order of 15-20 lb [5,600-7,500 litre (NTPD)] per man per minute. The opening of the discharge valve through which the air leaves the cabin is varied to a fixed cabin altitude-aircraft altitude schedule by a pressure controller. The controller is usually a pneumatic device utilising aneroid capsules and metal bellows. The output of the pressure controller to the cabin outlet valve is determined by the absolute ambient (pitot static) pressure and the absolute pressure in the cabin, and the control law. The control law is fixed and cannot be varied by the aircrew. The cabin pressurisation controls which can be operated by the pilot include an air shut off valve switch, operation of which shuts off the air flowing into the cabin, a cabin pressure dump switch that opens the cabin outlet valve fully, and a control that introduces external air into the cabin--the ram air facility. Often two of these controls are combined into a single control.

Present Cabin Pressurisation Schedules

The existence of two types of pressurisation schedules for cabins for low differential pressure cabin combat aircraft has been outlined in the introduction to this paper. With the schedule employed in the United States (17) (Figure 1), the cabin altitude equals the aircraft altitude [the absolute pressure in the cabin exceeds the ambient pressure by -0 to +0.25 Lb in^{-2}] at aircraft altitudes between ground level and 8,000 feet; the cabin altitude is held constant at 8,000 feet [10.92 ± 0.15 Lb in^{-2} absolute] at aircraft altitudes between 8,000 and 23,000 feet; and at aircraft altitudes above 23,000 feet the differential pressure between the cabin and ambient is held constant at 5.0 ± 0.15 Lb in^{-2}. The major characteristic of the pressurisation schedule employed in the United Kingdom (3) (Figure 1) is that the aircraft altitude at which pressurisation of the cabin commences is 5,000 feet and that the maximum cabin pressure differential (which is 5.25, 0 + 0.2 Lb in^{-2}) is only operative at aircraft altitudes above 40,000 feet. At aircraft altitude between 5,000 and 40,000 feet, the absolute pressure in the cabin varies directly with ambient (atmospheric) pressure. In practice, the two schedules yield very similar maximum cabin altitudes, e.g., a cabin altitude of 19,500-20,000 feet at an aircraft altitude of 50,000 feet. The major difference between the two types of schedule viewed aeromedically is that for a given rate of descent of the aircraft, the rate of increase of absolute pressure in the cabin is considerably greater (Figure 5) with the US schedule than with the UK schedule except, of course, between aircraft altitudes of 8,000 and 23,000 feet where the rate of change provided by the US schedule is 0. With high rates of descent from aircraft altitudes above 23,000 feet and at altitudes between 8,000 and 5,000 feet the US schedule requires more frequent venting of the middle ear cavities and with very high rates of descent would be expected to cause a greater incidence of otitic and sinus barotrauma than that associated with the UK schedule. The lower cabin differential pressure produced by the UK schedule at intermediate altitudes (10,000 - 30,000 feet) may well have airframe design advantages. The reduction of pressure over the upper surface of the aircraft which occurs at high speed is maximal at intermediate altitudes. The magnitude of the cabin differential pressure at these intermediate altitudes together with the suction over the upper external surface of the cabin determine the strength required of the cabin structure.

The UK type of pressurisation schedule (3) has been used in all the low differential pressure cabin combat aircraft developed for the Royal Air Force since the late 1940s, though with lower maximum differential pressures depending upon the altitude ceiling of the aircraft. It was also adopted for the tri-national Tornado aircraft. The four nation Eurofighter aircraft has, however, the standard US pressurisation schedule.
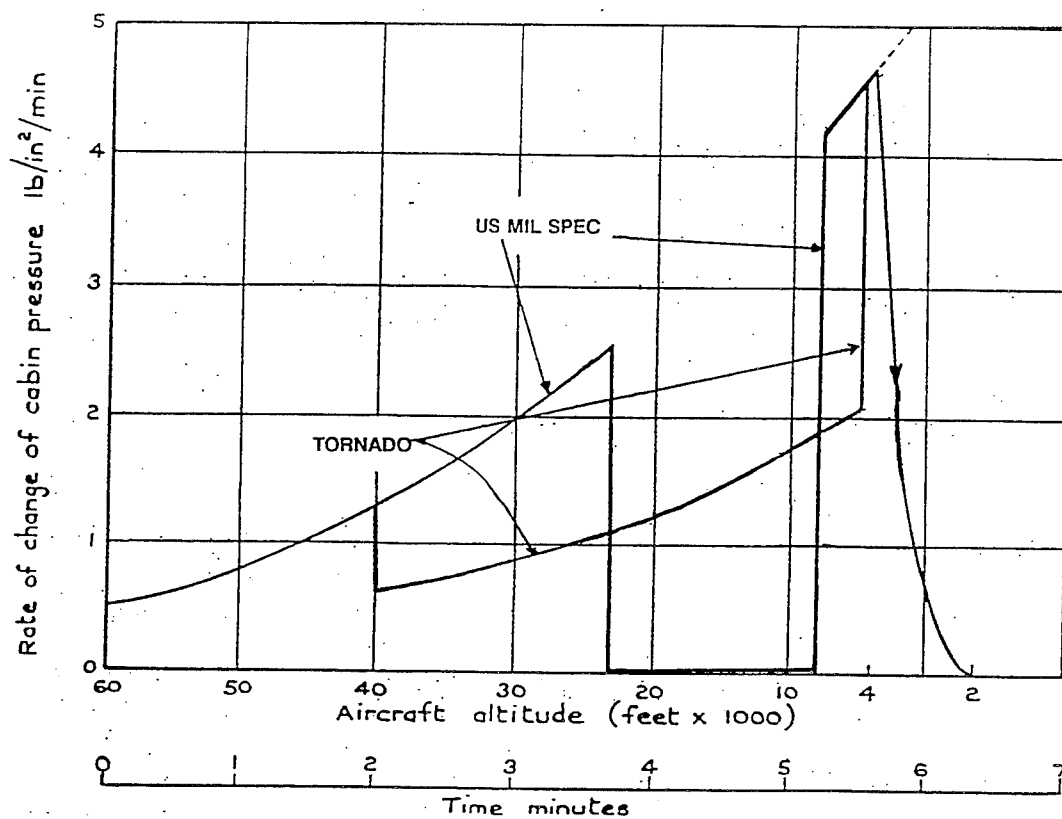
Figure 5. Comparison of rate of increase of absolute pressure, for a given rate of descent, US and UK.

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Cabin Pressurisation Schedule for Future Combat Aircraft

There are strong operational requirements for the new generation of fighter aircraft to operate for considerable periods of time at altitudes significantly above 50,000 feet. Current cabin pressurisation schedules which employ a maximum cabin differential pressure of 5.0 - 5.25 Lb in^{-2} will not maintain an acceptable cabin aircraft at those aircraft altitudes. Thus the cabin altitude will rise to 22,500 feet at 60,000 feet and to 24,000 feet at an aircraft altitude of 70,000 feet. These cabin altitudes are significantly above the level at which decompression sickness will occur and they almost certainly increase the risk of serious decompression sickness arising rapidly following loss of cabin pressurisation. Aeromedically, it is extremely desirable that the cabin altitude in these aircraft does not exceed 18,000 feet at the aircraft ceiling and indeed Webb et al. (19) have argued that it should not exceed 16,000 feet, in order to reduce the risk of venous gas emboli. The maximum cabin differential pressure in new high-altitude fighter aircraft should be increased above 5 Lb in^{-2} so that the cabin altitude at the aircraft's operational ceiling does not exceed 18,000 feet. This maximum differential pressure is only required at aircraft altitudes above 40,000-45,000 feet, and there may well be structural, and hence aircraft mass, advantages in employing a UK type of pressurisation schedule which, whilst minimising the rate of increase of cabin pressure on rapid descent, also reduces the magnitude of the cabin differential pressure at aircraft altitudes up to 40,000 feet. Typical examples of such pressurisation schedules are presented in Figure 6.
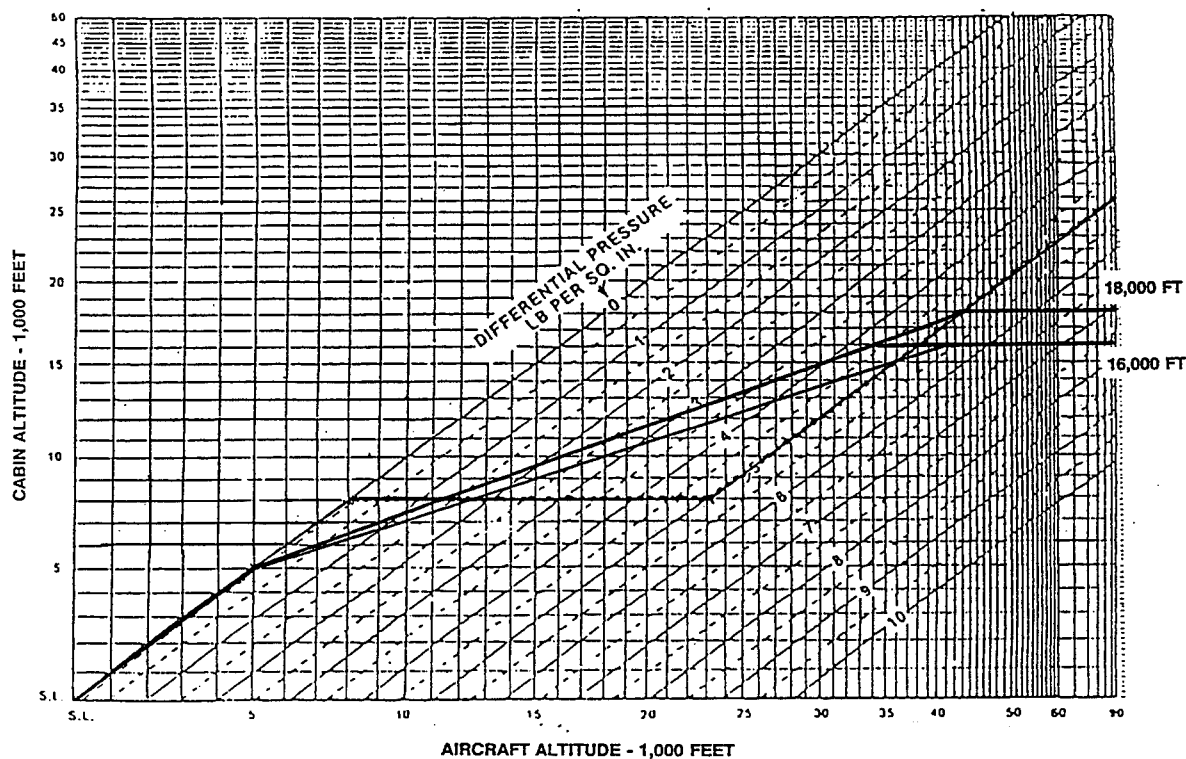
Figure 6. Typical examples of such pressurisation schedules.

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HIGH-ALTITUDE PRESSURE PROTECTIVE EQUIPMENT:
A HISTORICAL PERSPECTIVE

William J. Sears, Colonel, USAF (Ret), Ph.D.

Introduction

Aircraft engine/airframe technology and mission threats are evolving at such a rapid pace that by the year 2005 and beyond, it can be expected that aircraft will be capable of flying much higher and faster, withstanding higher G-loads, maneuvering rapidly in all axes, and remaining aloft longer. These advancing engine/airframe technologies coupled with increasing threats will expand future mission profiles and thus increase requirements placed on aircrew. These factors make it even more imperative in the future that aircrew performance and protection be an essential element of initial aircraft design and development. Although many of the potential man-centered problems have been identified and solved over the past half century, it remains to enhance the design of the man-machine interfaces to achieve reliability and flexibility, and the life-support equipment to assure protection and increased performance capabilities.

This paper provides a review of high-altitude pressure protective life-support equipment. The review is intended to present a thumbnail sketch of the development and use of a variety of pressure-protective ensembles and ancillary equipment that have been used over the years, as well as provide the basis for a re-examination of pressure systems that will allow the crewmember to be safely exposed for considerably longer periods at 60,000 feet and above.

Background

Mission Threats

There is a diversity of hostile Nuclear/Biological/Chemical (NBC) and newer directed-energy systems that can be, or are soon to be deployed (1). Indeed, it is realistic to assume that the future combat pilot will be subjected to nearly all of the same weapons as the combat soldier. These threats, which are especially hostile at lower altitudes, include:

- CHEMICAL/BIOLOGICAL WEAPONS--Artillery, rockets, missiles, bombs/bomblets, spray, vectors and covert sabotage.

- NUCLEAR EFFECTS--Fallout, blast, heat/flash and radiation EMP.

- DIRECTED ENERGY WEAPONS--Low-energy laser weapons, high-energy lasers, microwave weapons, target designators and range finders.

Newer Protection Requirements

Physiological limitations of the aircrew have always been a problem in military aircraft. Until recently, however, the aircrew has been able to exploit the full performance potential of operational aircraft. The following listing summarizes the changes required in crew protection forced by the newer mission threats (1):

- HIGH-SPEED AND LOW ALTITUDE FLIGHT--There is a greatly increased need for higher maneuvering Gz (terrain following and avoidance, missiles, directed-energy weapons, guns, and aircraft avoidance), and protection from windblast upon ejection (increased Q forces), greater ejection forces, and potentially increased exposure to NBC (low-level penetration). No change is required in altitude protection.

- HIGH-SPEED AND HIGH-ALTITUDE FLIGHT--There is little change in maneuvering Gz (low air density), windblast upon ejection (low KEAS) and NBC protection. There will be an increased need for high-altitude protection systems.

Protection Systems

A variety of protection systems have evolved over the years. "Enclosed seat" concepts including canopy seats, ejectable pods, crew modules and separable forebodies have received intermittent attention since the early 1950s. Only four enclosed systems were ever flight-qualified for air operations: the B-58 capsule, the experimental XB-70 ejectable pod, the F-111 crew module and the experimental B-1A crew module. Of these, the aircrew of the B-58 Hustler were approved for flight to 60,000 feet with only a pressure demand mask to provide oxygen upon decompression, i.e., no pressure vest or anti-G trousers. All crewmembers were trained to immediately close the capsule, which would automatically pressurize to 37,000 feet with a few seconds. However, assessments of development risks, weight, cost, producibility and advantages versus disadvantages have generally led to the development of a whole range of individually fitted pressure ensembles as protection against exposure to altitudes above 50,000 feet.

	PROS	CONS	COST
CAPSULES	SHIRT SLEEVE	EXTREMELY COMPLEX	VERY HIGH
	EFFECTIVE AT HIGH Q	HIGH WEIGHT	
	EASY ON CREWMAN	POOR EJECTION ENVELOPE	
		LOWERS A/C PERFORMANCE	
		HIGH DEVELOPMENT RISK	
		INCREASED VULNERABILITY	
CANOPY	EXISTING TECHNOLOGY	MEDIUM TO HIGH WEIGHT	MEDIUM
SHROUD		MARGINALLY EFFECTIVE	
SHIELD		SUIT PRESSURE AND	
		BREATHING SYSTEM REQ'D	
PRESSURE	LOW WEIGHT/MASS	Q FORCE LIMITED	LOW
SUIT	LOW VULNERABILITY	SUIT PRESSURE AND	
	HIGHLY EFFECTIVE	BREATHING SYSTEM REQ'D	
	OPTIMUM EJECTION		
	ENVELOPE		
	HIGHER-G EJECTION		
	HIGH ALTITUDE PRESSURE ENSEMBLES		

The first recorded suggestion for the use of pressure suits was by J. S. Haldane in 1920 who stated, "If it were required to go much above 40,000, and to a barometric pressure below 130 mm Hg, it would be necessary to enclose the airman in an air-tight dress, somewhat similar to a diving dress, but capable of resisting an internal pressure of, say, 130 mm of mercury. This dress would be so arranged that even in a complete vacuum the contained oxygen would still have a pressure of 130 mm Hg. There would then be no physiological limit to the height attainable.".

Since military application was limited in the early years, efforts involving high-altitude protection were generally left to adventurers and their scientific advisers. Early pressure suit development flourished as a result of both aviation and balloon contests: England (late 1933); USA (April 1934); USSR (late 1934); France (June 1935); Germany (late 1935); Spain (1935); Italy (1936); Canada (1942) and; Japan, Poland, China, Norway, Sweden and Czechoslovakia (1943).

Over the years a multitude of developmental and operational pressure protection systems has been produced. The following listing is an attempt to recall many of these systems in the anticipation that other individuals who have been intimately involved in the development of early prototype or operational pressure suits will critically review this information and add their input to these recollections. Most of the listed developmental and production pressure suits naturally evolved as attempts to provide a, "more comfortable, lightweight, and functional protective system," that conformed to the requirements of specific operational conditions, e. g., from short term exposure to altitudes above 50,000 feet to moon walks in a vacuum.

Pressure Suit Chronology

1933   First full-Pressure Suit--English firm for American balloonist Mr. Mark Ridge-Suit taken to 17 torr (84,000 feet) pressurized to 36,500 feet.

1934   Wiley Post Suit--B. F. Goodrich, full-pressure suit of double-ply rubberized parachute fabric, pigskin gloves, rubber boots, aluminum helmet, pressurized to 7 PSI, 10 flights before Post's death in 1935.

1934   In USSR Pressure Suit Developments--See: "Soviet High-Altitude Pressure Suit Development, 1934-1955", by Wilson, Charles L., Aerospace Medicine, Vol. 36, No. 9, September, 1965; "Origins of Soviet Space Pressure Suits, 1930-1963", by Shayler, David J., Journal of The British Interplanetary Society, Vol. 43, pp. 417-423, 1990 and; "Space Suits, Concepts, Analysis and perspectives", by G. Severin, Zvezda, June 1990.

1935   English broke two world records with the Mark Ridge Suit.

1935-45   Germans designed a full-pressure suit of laminated silk and rubber, reinforced net of silk cord, several models, covered with metallic outer covering to prevent ballooning-11 PSI without "sacrificing mobility" but very heavy. By the end of the war they used a separate oxygen-breathing and suit-gas system.

1935   French designed full-pressure suit, Drs Rosensteil and Garsaux, with backing of Potex Airplane Company.

1937   An Italian suit was taken to 51,000 feet.

1941   United Kingdom, Canada and USA studied the use of pressure breathing mask, vest and inflatable trousers for short-term high-altitude protection.

1943-46   Henry, et. al., at the University of Southern California designed the capstan partial-pressure suit and exposed subjects to 80,000 feet--3 models.

1946-48   David Clark Company developed Dr. Henry's original capstan partial-pressure suit and produced the first operational models in custom sizes for early rocket-powered X-Plane test pilots, e.g., Yeager et. al.

| 1947 | Most of the emphasis in the newly formed USAF was directed towards partial-pressure suits while the USN placed their emphasis on omni-environmental full-pressure suits to combine altitude and immersion protection. |

1947 Most of the emphasis in the newly formed USAF was directed towards partial-pressure suits while the USN placed their emphasis on omni-environmental full-pressure suits to combine altitude and immersion protection.

1948-53 T-1 Capstan Pressure Suit--The first operational capstan partial-pressure suits (PPS) were produced in custom sizes for early rocket-powered X-Plane test pilots, by the David Clark Company. They produced the T-1 capstan pressure suit in standardized sizes made of nylon/ cotton twill. It was chamber tested to 106,000 feet and subsequently flown in a variety of high-altitude aircraft. The T-1 Capstan suit (5-to-1 ratio), incorporated anti-G suit, no chest bladder, 12 standardized sizes for fighter aircraft. David Clark Company.

1948 Mark 1 Mod III Suit--Omni-Environmental Full-pressure Suit--USN development; many modifications over 10 year period, B.F. Goodrich. Suits had earlier been developed by Goodrich for the Doolittle mission (1942).

1950 Model 4--A Full-Pressure Suit developed for D-558-2 Douglas Skyrocket test pilots. First Flown by Navy test pilot Marion Carl for 85,000 feet altitude record flight. Integrated arm scye bearings (non-sealed) for improved mobility. Custom sizing. Forerunner of X-15 full-pressure suits. David Clark Company and Scott Crossfield, NACA.

1950s MC-2--Full-pressure suit with integrated parachute harness, first used in the X-15 aircraft, custom sizing, many variants. David Clark Company Model S794-5.

1950s RAF Jerkin System-pressure vest used with P/Q mask and anti-G suit. Several variations including unsleeved, sleeved and integrated garments proven for short-term protection to 60,000 feet. Mk-1, Taylor and M.L. clam-shell pressure helmets used with sleeved variant for short-term protection to 100,000 feet.

1950s Canadian Waistcoat-Mask/Vest/G-Suit partial-pressure assembly. Canadians studied variants of this assembly as far back as the early 1940s. Newer variants have been studied for short term protection to altitudes of 80,000 feet with G suit pressurized to 4 times breathing pressure.

1953-54 S-2--Modified capstan partial-pressure suit evolved from the T-1 with no anti-G, no chest bladder, 12 sizes for bomber aircraft. Used K-1 helmet, A2 adapter, C-1 assembly with F-1 regulator (53 cubic inch bottle at 1800 PSI) for oxygen source. Fired automatically by aneroid at 43,000 feet. Used T block to hook aircraft oxygen system with C-1 assembly backup. Hawks pressure compensated valve in K-1 helmet assembly. David Clark Company.

1955 S-4--A modified S-2 partial-pressure suit, no anti-G, chest bladder incorporated for ease of breathing. Also incorporated abdominal bladder for individuals who experienced abdominal difficulties, e.g. weak inguinal rings.

1956 MC-1--A modified S-2 partial-pressure capstan suit with chest breathing bladder, 12 sizes, high altitude, fighters and bombers, smaller capstan in torso area, pressure gloves, K-1 or MB-5 helmet. David Clark Company.

1957-63 MB-1 & 2--Experimental test pilot's partial-pressure suit, chest and abdominal bladders added to G4A anti-G suit for Air Defense Command, provided protection to 65,000 feet, no capstans, used with MA-1 helmet rather than the K-1. David Clark Company.

1957 MC-3--A capstan partial-pressure suit with horizontal shoulder zipper, sewn breaklines, no anti-G, height/weight sizing criteria, bomber and reconnaissance aircraft, 12 sizes. David Clark Company. MA-2 helmet by ILC Dover. Many variants of this suit for special projects, e.g., Yeager et. al., SX-1,

X1A, X-2 aircraft, Simons et. al., Project Man High balloon flights and Kittinger's Project Excelsior stratosphere parachute jump from 102,800 feet, 1960.

1958 C-1A--A partial-pressure capstan suit with incorporated anti-G bladders for USN fighter aircraft, 12 standard sizes. David Clark Company.

1958 MC-3A--A modified MC-3, vertical shoulder laces and adjustable break lines, David Clark and Berger Brothers. MA-2 helmet by ILC Dover.

1958-64 MC-4--A partial-pressure capstan suit, vertical shoulder laces, adjustable break lines, anti-G suit bladders, MG-1 (Berger Bros.) gloves, MA-2 helmet by ILC Dover. Suits by David Clark and Berger Brothers, adapters by Airlock, seat kit and oxygen panel by Firewell, 12 sizes.

1958-64 MC-4A--A modified MC-4, height/weight fit for fighter aircraft, anti-G suit. Suits by David Clark, Berger Brothers and Seymore Wallace. The MC-3 and MC-4 series of suits used the MB-5, MA-2 (ILC Dover) and MA-3 (Bill Jack) helmets.

1958 CSU-2P--Developmental dual capstan partial-pressure suit for altitude protection by Berger Brothers. Used pressure socks and double capstan for looser fit.

1958 Lombard Suit--Developmental Partial-pressure suit developed by Dr. Lombard of Northrop.

1960 Mercury Suit--Modified USN Mark IV Series full-pressure suit produced by B.F. Goodrich. The custom suit was developed as back-up emergency systems for intra-vehicular activity (IVA).

1960s CSU-4/P--A Bladder type partial-pressure suit, quick don, 8 sizes, separate garment (CWU-4/P) for immersion protection, inverted neck seal, HGU/8/P helmet, front entry, get-me-down altitude protection, no ventilation initially, gloves needed for longer term exposure to high altitude. Used by Kittinger in USAF stratosphere jump, later used by Japanese. David Clark Company.

1960s CSU-5/P--A modified bladder type partial-pressure CSU-4/P suit, integrated wet suit. David Clark Company.

1960s A/P 22S-3--USAF adopted USN Mark IV suit development (B. F. Goodrich and Arrow Rubber Company). Full Pressure, 2 layers, oxygen regulator exterior of helmet, 12 torso sizes, 7 gloves sizes, 2 helmet sizes, pressure relief set in the 3.5 to 4.0 PSI range, material used in mobility areas was Helenca coated neoprene, in non-mobility areas was nylon impregnated chloroprene.

1960s A/P 22S-2--Greater mobility than the A/P22S-3 full-pressure suit, 4 layers, 8 sizes, suit controller, oxygen regulator inside helmet, outer layer nylon/polyurethane, dacron link net restraint second layer, third layer was silicon impregnated nylon/neoprene pressure bladder, inner fourth diffusion layer was oxford weave. Many variants, used in bomber aircraft, the X-15 and other high-altitude aircraft. David Clark Company.

1960s S-1029--Developmental bladder type partial-pressure suit. David Clark Company.

1960s TFX--Prototype bladder type partial-pressure suit with a separate Anti-G suit valve. APL program with Navy and ILC Dover.

1960s AES Series--Hybrids using laminated fabrics, rolling convolutes, toroidal joints, sealed bearings, modular sizing. AiResearch and Litton Companies.

1960-80s   S-100--Many modifications from early MC-3A capstan suits. Torso bladder using redundant full pressure controller and full pressure helmet (1972), 12 sizes, high-altitude reconnaissance aircraft, exterior cover of various colors worn over pressure suit. Last capstan partial-pressure suit in operational service--retired with U-2C aircraft in 1989. David Clark Co.

1960s   Type B--Full-pressure suit designed by R. E. Simpson, and developed by Baxter, Woodhouse and Taylor Ltd. for the Royal Air Force. Used Windak full pressure helmet or lightweight head enclosure (handbag) developed by the Royal Aircraft Establishment.

1960-70s   Swedish Jerkin--Partial-coverage garment--two pressure flying suit with diaphragmatic bladder, used with high-pressure mask equivalent to A-13 with Hardman kit. Anti-G suit at 3.2 times breathing pressure, get-me-down protection to 65,000 feet. Mask automatically tightened by gas filled expansion pad in back of helmet.

1960-80s   French EFA-30--Partial-pressure capstan suit using full pressure buffet protective helmet.

1960-62   SPD-117--Prototype full-pressure suit for USAF Manned Orbital Laboratory definition, thermal garment and PLSS by ILC Dover and Firewell Companies.

1960-70s   S901/970--A-12, YF-12A and SR-71 full-pressure suit with integrated subsystems, parachute harness, automatic flotation system, urine collection device, redundant pressure control and breathing system, thermal protective garment, Custom plus 12 sizes, various models. David Clark Company.

1960-70s   S1010--Special projects full-pressure suit with integrated subsystems including parachute harness, automatic flotation, redundant pressure control and breathing system, thermal protective, Custom plus 12 sizes, various models used in U2-R. David Clark Company.

1962   Macuh Suit--Closed cell foam suit concept by Macuh Laboratories, USAF/NASA study, report MLTRD-62-13.

1962   Lines of non-extension suit--Developmental partial-pressure suit concept by Rand Corp.

1962-66   Gemini suit--NASA Gemini full-pressure suit, closed loop, G-1C, G-2C, G-3C (IVA suits), G-4C (both IVA and EVA suit) used for first U.S. spacewalk by Ed White, Gemini 4, June 1965. The G-5C incorporated a soft head enclosure for the 14-day Gemini 7 mission, Borman and Lovell, IVA. David Clark Company.

1962-64   S-939--Full-Pressure Suit for X-20A Dyna-Soar program. David Clark Company

1963-65   A4H--ILC Dover and Hamilton Standard full-pressure suit, Contained a secondary bladder and restraint with a wrist cuff/dam for NASA/HSD (1963-1964), modified A4H suit for NASA-AMES (1964-1965).

1964-65   AX5L--NASA Apollo suit prototype, IVA, ILC Dover

1964-68   RX-Series--RX-1 Litton hardsuit full-pressure suit-weighed 90 pounds, rolling convolute joint technology, 2-plane enclosure, modular sizing, 1964. RX-2, 90 lb. in 1964. RX-2A, 80 lb. in 1964. RX-3 and RX-4 down to 60 lb. in 1966-pressurized to 5 PSI but adaptable to 7 PSI with oxygen/nitrogen mixture. The RX-5A was the final configuration of this series.

1964-80s   AX-Series--Between 1964 and 1968 two hard-suit assemblies were developed at NASA-ARC, identified as the AX-1 (Ames Experimental) and AX-2. These suits were first to demonstrate multiple bearing technology. The AX-3 was an 8 PSI suit, 50 pounds, 7-10 PSIG operational

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pressure, with improved mobility and was completed in 1977. The program culminated in the development of the prototype AX-5, an all-hard suit for high-pressure application and zero prebreathe in the 1980s.

1965 A-1C--Full-pressure suit, closed loop system, custom sizing, Apollo Block I Program. David Clark Company.

1965 AX-1C--Full pressure, Apollo Block II, IVA/EVA. David Clark Company.

1966 Boyles Law Suit--Concept by Otto Schueller, patented by Davis, Moore, Ritzinger and Whitmore at USAFSAM. Passive suit pressurization bladders containing closed gas cavities and breathing bladder connected through helmet regulator and aneroid operated visor. Studies to 75,000 feet using system. Further developed by David Clark Co.

1966 MD Series--MD1, MD2, MD3, and MD4 full-pressure suits, closed loop system, 12 sizes projected for USAF Manned Orbital Laboratory definition program. David Clark and Hamilton Standard Companies.

1967 A/P22S-4--Full-pressure suit replacement for A/P22S-2, 8 sizes, bomber and reconnaissance aircraft. David Clark Company.

1967-69 A7L--Developmental NASA Apollo suit. The A6L was the precursor to the A7L and was never flown. A7L suit was developed and used for IVA-Apollo 7 through 14. Lunar configuration designed for EVA. ILC Dover.

1970s A/P22S-6--Full-pressure suit replacement for A/P22S-4, 12 sizes, bomber, reconnaissance and fighter aircraft. David Clark Company.

1970s A/P22S-6A--Modified A/P22S-6 suit to add urine collection device with other material and hardware changes.

1970-73 EIS/OES--Developmental 8.0 PSI Emergency Intravehicular Suit (EIS) and Orbital Extravehicular Suit (OES) programs. Fabric mobility joint technology base for Shuttle EMU development.

1970-75 A7LB--NASA Apollo Suit designed for both IVA and EVA, used for Apollo 15 through 17. A7LB with thermal garment used for skylab. ILC Dover.

1970-80s S1030--Upgraded SR-71 full-pressure suit, link net with integrated subsystems. David Clark Company.

1975-94 Shuttle EMU--Hamilton Standard and ILC Dover modular suit for Space Shuttle. Many variants over the years. Eight EVA certification, modularized to fit 90% male/female population. Integrated hard torso with portable life support system, RF sealed bladder, fabric mobility joint elements, waist bearing, improved pressure gloves.

1977 PHAFO--Prototype High-Altitude Flying Outfit. Prototype partial-pressure suit by David Clark to integrate altitude, thermal, immersion, chemical defense and anti-G protection, Non-conformal (Dome Type) full pressure helmet with oxygen mask.

1977 HAFO--High-Altitude Flying Outfit. Prototype developmental full-pressure suit with integrated thermal/pressure/chemical defense/immersion and Anti-G protection. ILC Dover.

1977   HAPS--High-Altitude Protective System (HAPS). Hybrid get-me-down system assembled for NASA Dryden Flight Research Center test pilots. Consisted of Cut-away Anti-G suit with dual bladders (altitude and G), torso counter pressure garment (Jerkin) and British P/Q type oxygen mask. Breathing pressure to 70 mm Hg at 60,000 feet. Combined effort among DFRC (Barnicki), Edwards AFB (Melvin), RAFIAM (Ernsting), USAFSAM (Morgan), and David Clark Company.

1979-82   S1030A--NASA Shuttle Orbital Flight Test Ejection Escape Suit (STS1-4). Modified S1030 with anti-G suit system. David Clark Company.

1980-90s S1031--Special projects full-pressure suit. Updated version of S1010, 12 sizes, used in TR-1 and U2-R, David Clark Company.

1980   TR-1--Prototype full-pressure suit developed by ILC Dover for TR-1 aircraft.

1980s   MK 3 ZPS--NASA Zero pre-breathe full-pressure suit developed to preclude need for denitrogenation prior to EVA. The Mk-I ZPS was the precursor to the Mk-3 and was based on shuttle suit geometry with several rigid components with use pressure at 8.3 PSI. The MK-3 contains both machined/cast aluminum and composite graphite Hard Upper Torso and modular sections for arms and legs to fit 90% of male and female population. Uses rolling convolutes at shoulder, and waist; bearings in hips; fabric elbows, knees and ankle joints. ILC Dover, Airlock Corp. and Hamilton Standard.

1982   TLSS/ALSS--Tactical Life Support System. Developed by the USAF and Boeing/Gentex et. al. to provide get-me-down protection from 60,000 feet. Incorporated many new features for a modular mask, vest, anti-G suit ensemble integrated to provide PBG for high-G maneuvers and PBA for altitude with G trousers providing 4 times the breathing pressure from a molecular sieve oxygen concentration system. There are now many variants of similar protective design in the United Kingdom, Canada, Sweden, and an Advanced Oxygen System from France.

1987-89   AHAFS--Advanced High-Altitude Flight Suit. High pressure (5-6 PSI) full-pressure suit developed for the USAF to increase mobility at higher operating pressures. ILC Dover.

1987   Shuttle Launch/Entry Suit--Combination pressure/immersion modified CSU-4/P suit with non-conformal full pressure helmet, dual neck dam, integrated exposure suit, parachute harness and flotation equipment for emergency escape from shuttle to 100,000 feet. David Clark Company.

1989   APS--Advanced Pressure Suit (APS) bladder type partial-pressure suit designed and developed by Northrop and ILC Dover for F-23 Advanced Tactical Fighter. Integrated high altitude, Anti-G, Immersion, thermal air cooling and chemical defense protection. Suit bladder with pressure booties and oronasal/full head pressure for PBA and PBG. Breathing bladder/helmet/suit pressurized to 3.5 PSI for altitudes above 35,000 coupled with 60 mm Hg breathing/10.5 PSI suit at +9z for anti-G protection, 6 each purchased by USN.

1990s   ESA--Prototype full-pressure suit for the European Space Agency (ESA), produced by Dornier, Dassault, Zodiac, et. al., 6 PSI.

1990s   EVA 2000--Prototype full-pressure suit effort between ESA and USSR to upgrade the Orland DMA.

1990s   S1034--Prototype Special projects full-pressure suit to replace the S1031. Integrated life support subsystems to include breathable pressure bladders. David Clark Company.

1990s   S1035--NASA Shuttle full-pressure Advanced Crew Escape Suit (ACES) to replace the S1032 LES. Integrated life support subsystems to include breathable bladders. David Clark Company.

1993-94   Partial-pressure suit development for F-22 Aircraft. Get-me-down partial-pressure ensemble combining Mask/Vest/uniform pressure anti-G garment for protection to 60,000 feet. USAF contractors include Boeing, ILC Dover, META and Helmets Ltd.

1993-95   Space Station EMU--Upgraded version of the Shuttle EMU with improved sizing and mobility, 25-mission EVA certification. Hamilton Standard and ILC Dover.

Current Status of High-Altitude Protection Systems

Overview

Although full-pressure suits have been shown to provide long-term protection against many of the effects of high-altitude exposure, they characteristically have received only marginal acceptance among crewmembers of high-performance tactical aircraft. Impairment to mobility, comfort and general effectiveness preclude their application for many missions except high-altitude reconnaissance or flight-test missions. Their use often involved restrictions to the pilot's visibility, mobility and dexterity, which compromised mission effectiveness. Significant degradation of visibility is one of the most pervasive arguments against use of a pressure suit in high-performance aircraft. Although the visor may be free of distortion in critical visual areas, sortie success rate using an optical sight may be seriously degraded when aircrews are required to use pressure suits with the visor closed. Also, night refueling with a visor in front of the eyes is often difficult and can be dangerous. Additionally, the visor acts as a condensing lens and is especially disturbing when the sun is in the forward field of vision. All these problems, however, have once again arisen in the newer chemical defense and laser protective visor enclosures.

Partial-pressure suits range from the current mask/vest/anti-G suit systems, which provide only a few minutes protection at altitudes above 60,000 feet, to the earlier, more fully encapsulating capstan or bladder suit systems. These systems have been shown to provide several hours protection at altitudes above 60,000 feet, providing there was adequate prebreathing to prevent decompression sickness, e.g., MC-3 capstan suit(3 hours)and CSU-4/P bladder suit (2 hours)(2).

Current and Future Requirements

While it is nearly impossible to predict all new threat scenarios that may evolve over the next ten to twenty years, attempts to outline anticipated needs for high-altitude systems have been formulated for both full-and partial-pressure ensembles. It can be seen that many of the requirements overlap. For full-pressure systems associated with reconnaissance flights the following apply (3):

- Protection to altitudes above 80,000 for extended periods (16 hours).
- Ejection/windblast protection to mach 3.
- Protection against high and low temperatures.
- Protection against NBC agents and provides for self don/doff capability in the NBC environment.
- Provide a system that is lightweight, durable and easily maintained in an NBC environment, to include being decontaminatable.
- Provide adequate mobility for accessibility to aircraft and instruments without assistance.
- Eliminate requirement for prebreathing 100% oxygen with oxygenation equivalent to breathing air at sea level.
- Allow fluid intake, feeding, urine output, temperature regulation and increased tactile capabilities.
- Provide a lightweight helmet, optically correct visor with unrestricted vision, visor heating and defogging, anti-reflectance coating, improved spectacle mounting, communications and improved head mobility.

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- Provide anti-drown and anti-suffocation features.

- Provide laser and flash protection.

- Provide coverall with improved hold-down, integrated flotation, parachute harness, fire protection and ancillary hardware.

- Provide gloves with improved sizing, tactility, and dexterity.

- Provide built-in test features.

- Provide better supportability and reduce training with minimum service life of six years.

The requirements for <u>partial-pressure systems</u> vary considerably depending upon the perceived need for longer-term high-altitude protection. If it is assumed that near-term weapons will nearly close the window for low-and fast flight in the near future or flight at high altitudes is necessary to prevent immediate descent into the higher threat environments over the target area, the following requirements can be envisaged (4):

- Provide for primary protection to 70,000 feet for one hour or, at a minimum, provide protection to 55,000 feet for one-half hour after loss of cabin pressure to reduce the need after decompression to descend into a high-threat environment over the immediate target area.

- Provide for +9 Gz, -3 Gz and +/- 3Gy acceleration protection.

- Provide ejection protection to 710 KEAS (Q= 1728 psf) and buffeting loads to 25 ft-lb.

- Provide NBC protection for the anticipated mission duration, during egress from the aircraft and adequate filtration systems both to and from the aircraft and during the flight. Additionally, provide for safe don/doff of the ensemble with subsequent return to the flight configuration.

- Provide a cooling/ventilation system with anti-exposure protection.

- Provide a helmet assembly with anti-buffet protection, unassisted don/doff capability, removable/cleanable/sized-to-fit comfort liner, communication and oxygen system, cooling/ventilation system, anti-suffocation and anti-drowning feature, clear visor/sunshade and laser protection, integrated personal leads for oxygen/communications/defog and ventilation, vertical rise restraint, adequate head mobility, overpressure safety relief feature, gas tight integrity at suit assembly interface, test equipment attachment or penetration points, and component replacement capabilities.

- Provide a suit assembly that can be donned and doffed without assistance, with a removable and washable comfort liner, cooling and ventilation system, integrated torso harness for parachute attachment and survival kit connection, integrated anti-G/flotation/crewman to seat restraint and ballistic protection, NBC protection, urine elimination device, overpressurization relief, suit pressure monitoring, crewman adjustable comfort and pressurization controls, adequate mobility in pressurized state, fire resistant outer protective covering, redundant zipper protection against single point failure, sizing through 5th to 95th percentile, test equipment attachment points and component replacement capability.

- Provide gloves with improved sizing, tactility, and dexterity.
- Provide better supportability and reduce training with minimum service life of six years.

Conclusion/Recommendations

The newer Anti-G/Get-Me-Down systems are being designed to interface with NBC and laser visor protection coupled with Helmet Mounted Displays and Night Vision Systems. Indeed, the prototype COMBAT Aircrew Chemical Ensemble (ACE) system incorporates a fully enclosed helmet/visor/mask concept for anti-G and altitude protection to

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50,000 feet. It is only a step, albeit a fairly large one, to develop a system that would provide much longer-term protection to altitudes above 60,000 feet.

This would minimally include adding sleeves to the current vest, integrating the vest with uniform pressure trousers (ATAGS or equivalent), developing a full head enclosure and neck seal (a less robust system will be necessary for NBC protection), provision of pressure gloves (MG-1 or equivalent), and redesigning the oxygen regulator/controller system for both automatic and manual operation of the oxygen pressure schedule. All of these concepts have been prototyped at one time or another. Indeed, the late 1980s Northrop Advanced Pressure Suit was developed using the partial-pressure system requirements listed earlier providing both anti-G and long-term high-altitude protection (4). From lessons learned in these earlier studies coupled with newer materials and fabrication techniques, it is suggested that an improved, pilot-acceptable pressure helmet/suit, pressure and oxygen controllers, ancillary hardware and pressure gloves are well within the state-of-the-art.

The "pilot-acceptable" full head enclosure will present the most difficult challenge during development of an integrated high-altitude protective ensemble. One solution to the visual and pressure breathing for G problems associated with a full head enclosure is leaving the visor open during normal operations (non-NBC operations). This arrangement would require that supplemental oxygen be delivered to a high-pressure oronasal mask located inside the helmet similar to that in current PBG systems. The visor could be manually or automatically closed by a barometric sensing device coupled to the visor release unit. Indeed, you may recognize that such a helmet system was developed about 40 years ago by ML Aviation and others in the UK (5). A portion of the head crown was not covered by the bladder in this system, thereby balancing the pneumatic forces and subsequently avoiding the helmet lift. Such an innovative approach is currently needed to provide a stable platform for new helmet mounted visual displays and night-vision systems. If the USAF were to validate a need for an improved emergency pressure suit outfit, it is recommended that a variant of this concept be re-examined for possible use in a full head enclosure. This early configuration offered many desirable features that should enhance pilot acceptance and performance. A methodical, iterative program should be developed to provide such an integrated high-altitude protective system for future high-performance aircraft. It is important that such a program be initiated early to ensure that the technology will be available when required in the future.

Acknowledgements

I would like to thank the following individuals who have provided a most welcome editorial review of the chronological pressure suit data herein: (1) Joe Ruseckas, John Flagg and Jack Bassik, from the David Clark Company, (2) George Durney, Bob Wise (formerly with B. F. Goodrich) and Dave Cadogan, from ILC, (3) Joe Kosmo from NASA/JSC, (4) LTC Tom Morgan from the Armstrong Laboratory, Brooks AFB, Texas, (5) Colonel Tom Workman, Tom Bowen and Dick Cook from the 9th Medical Group/SGT, Beale AFB, California, and (6) Arnie Moore from Rothe Development Inc.

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4. ILC Dover, "Advanced Protective Design Study", Project 162 for Northrop Aircraft Division, 20 December 1984.

5. Bowen, J.D., Test Report on the ML Partial Pressure Helmet Compatibility with Boyles Law Emergency Pressure Suit", AMRL, Wright Patterson AFB, Ohio, August 1971.

Discussion

DR. SEARS: A CSU-4/P partial-pressure suit variant was developed as the emergency escape ensemble for NASA. Dr. Bomar, Capt. O'Connor and others were exposed in this suit to very high altitudes for a fairly long period of time. Would you tell the gathering what altitude you were exposed to, Dr. Bomar?

DR. BOMAR: It was 100,000 feet.

DR. SEARS: 100,000 feet, for half an hour?

DR. BOMAR: Forty minutes.

DR. SEARS: Forty minutes. So this gives you an idea that a partial-pressure assembly would allow an individual to remain at very high altitudes for at least 30 minutes under emergency conditions.

Use of Variable Oxygen Concentrations to 50,000 Feet

Robert B. O'Connor, Capt, USAF, BSC

Introduction

This presentation is based on a study called EONS that was completed several years ago. EONS stood for Expeditionary Oxygen Nitrogen System. It was a U.S. Navy system that, like aircraft on-board oxygen generation systems (OBOGS), enriched the oxygen concentration of air by selective adsorption of nitrogen on zeolite molecular sieves. Also like OBOGS, EONS produced a maximum oxygen concentration of 93-95%. For logistic and long-term cost advantages, EONS and OBOGS product gas was meant to replace the standard 99.5% aviator's breathing oxygen (ABO) produced by liquid oxygen systems. Consequently, it was necessary to investigate whether the decreased maximum oxygen concentration would present any dangers of hypoxia if used throughout the flight envelope. Since there was no reason to expect any hypoxia problems from EONS product gas at normal cabin altitudes, efforts concentrated on the worst case scenario of a rapid decompression to the emergency flight ceiling of 50,000 feet.

Methods

A search of the literature produced no record of an evaluation of the performance of the standard USAF breathing system at 50,000 feet when supplied with 99.5% oxygen. Therefore, that condition was investigated as a baseline measurement. Trials were also completed with 93% oxygen to represent normal OBOGS product gas. Ninety percent oxygen was tested to simulate product gas from a slightly degraded OBOGS and, finally, 85% oxygen was used to represent breathing from a significantly degraded OBOGS.

Comparisons of the four breathing gases were based on end-tidal oxygen partial pressures following a one-second, 5 psid rapid decompression (RD) from 20,000 to 50,000 feet. Tests were conducted with the breathing regulator in both the dilution and non-dilution modes.

Seventeen volunteer subjects took part in the study. However, nine of them either completed only a portion of the experimental conditions or had problems with data collection during one or more of their exposures. The most common source of trouble was significant mask leakage due to positive pressure breathing (PPB). Consequently, the data analysis for this report is based on eight subjects. All of them were male active duty Air Force service members ranging in age from 20 to 45 years old. All were experienced in altitude experiments and received refresher training at ground level in positive pressure breathing.

Life-support equipment used for all of the altitude exposures consisted of the following: the CRU-73/A oxygen regulator, designed to provide up to 30 mm Hg of positive pressure breathing at 50,000 feet; the MBU-12/P oxygen mask and HGU-55/P helmet; the CRU-60/P connector; standard 7/8 inch internal diameter oxygen hose; and a CSU-13B/P anti-G suit. The G-suit was not connected to a G-valve. The only inflation of the bladders came from expansion of any gas that was in the suit during the decompression.

The subjects were seated in a specially-designed support chair in a hypobaric chamber for all experiments.

Variables recorded during the trials were: heart rate; mask cavity pressure; inspiratory flow; carbon dioxide and oxygen concentrations measured by mass spectrometer; and O2 saturation (using an Ohmeda Biox III fingertip oximeter).

Figure 1 provides a graph of the altitude chamber exposure profile for all of the experiments. Each altitude exposure was preceded by sixty minutes of breathing 100% oxygen at ground level to reduce the risk of

decompression sickness. During this period, subjects completed a dual performance test. This consisted of a short-term memory task and a tracking task. Next was a 5,000 feet ascent/descent to check for ear or sinus problems. This was followed by an ascent to 27,000 feet to allow for the elimination of abdominal gas. The chamber then descended to 20,000 feet for baseline recordings and completion of a second set of performance tasks. It was at this point that the breathing gas feeding the subject's regulator was switched from 100% oxygen to a 93%, 90% or 85% oxygen mixture, depending on the experimental condition called for on that day. The subjects were unaware of the composition of the gas they were receiving. Also, for dilution tests, the regulator was changed to the normal or dilution setting. Three to five minutes were allowed for equilibration of the subject's blood and lungs with the test gas. Just prior to the decompression, the subjects were given a warning and instructed to breathe normally. Shortly thereafter, the chamber was decompressed from 20,000 to 50,000 feet in 1.0 to 1.5 seconds. Ten seconds after the decompression, the subject began a repeat of the performance task. After 60 seconds at peak altitude, the chamber was recompressed in approximately 15 seconds to 40,000 feet. Exposure at this altitude was terminated when the subject completed the performance task begun at 50,000 feet. Total time at or above 40,000 feet was on average three minutes. The final altitude stop was at 20,000 feet, where again a performance task was completed. This was followed by descent to ground level.
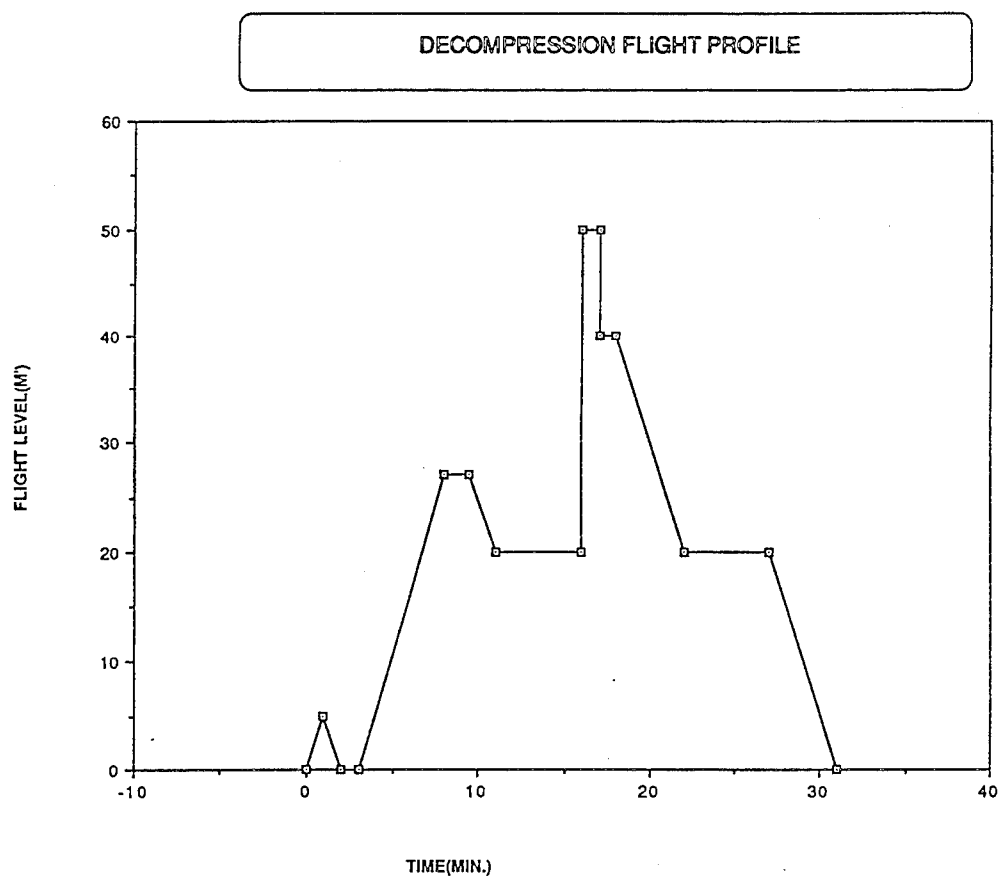
Figure 1. Decompression Flight Profile.

Results and Discussion

Figure 2 shows the end-tidal partial pressures of oxygen over time for the four test gases when the regulator was set in the non-dilution mode. The time course is 50 seconds before and after the rapid decompression, shown at time=0.

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Prior to the RD, when the subjects were breathing an undiluted gas mixture, all of the partial pressures of oxygen measured in the mask cavity were 225 mm Hg or greater. During the decompression, at time=0, the partial pressures for all the gas mixtures fall rapidly due to the sudden decrease in chamber pressure.
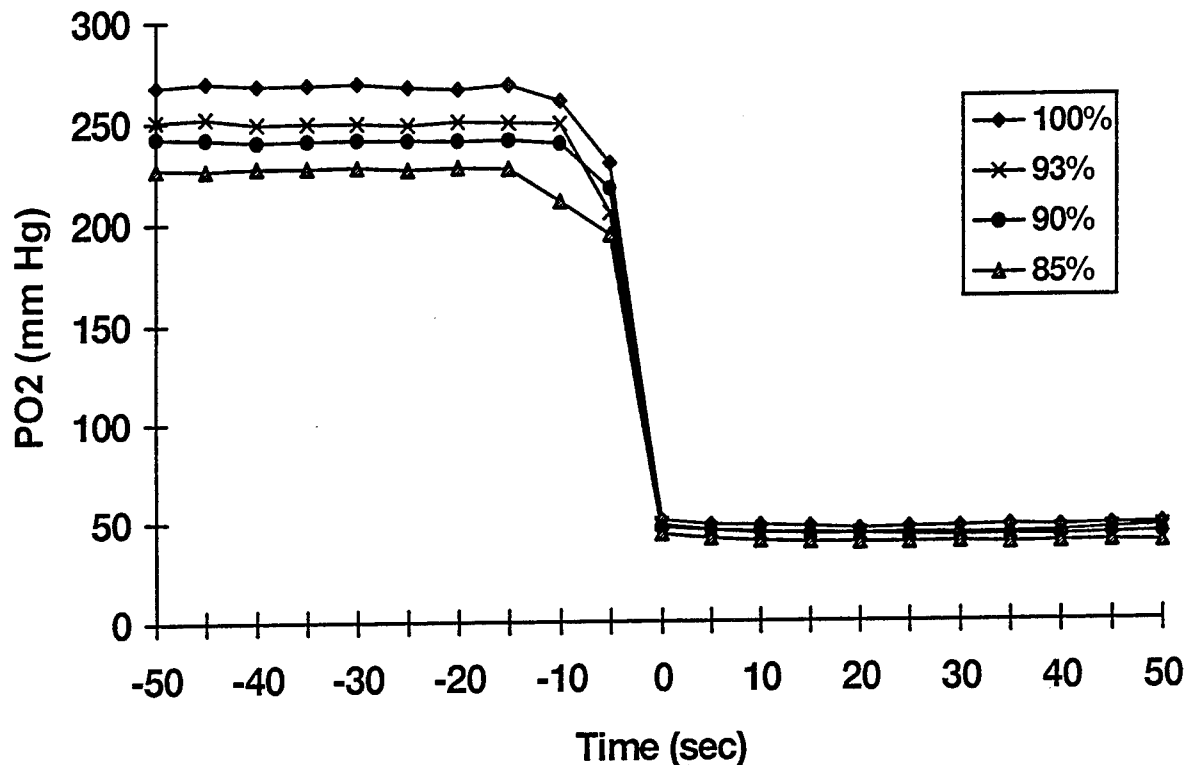
Figure 2. Mask Cavity Gas Concentrations vs. Time, Nondilution.

Figure 3 is taken from the previous figure and expands the graph for the time during and after the RD. The values every 5 seconds represent an average value for the 8 subjects at each 5 second interval. The four gas mixtures follow the same descending order that was evident before the RD. While none of the oxygen partial pressures for the four breathing mixtures fell below the 30 mm Hg limit recommended for post decompression protection against transient hypoxia, it's obvious that the margin of safety decreases with any degradation of the OBOGS product gas. It should also be mentioned that the breathing regulator used for these tests performed at the high end of the specification for positive pressure breathing (PPB) for altitude protection. It's possible for a regulator to deliver lower levels of PPB and still remain within specification. That scenario, or a significant leakage of gas from the mask, which frequently occurred, could reduce the margin of protection regardless of the oxygen concentration of the breathing gas.

The mean values of oxygen partial pressure for each of the gas mixtures in the non-dilution trials, averaged over the time at 50,000 feet, were as follows: 100% oxygen - 47 mm Hg; 93% oxygen - 44 mm Hg; 90% oxygen - 43 mm Hg; and 85% oxygen - 39 mm Hg. The 93% and 90% partial pressure values were significantly different from both the 100% and 85% results, but not from each other. The decrease in partial pressure for 85% oxygen versus the other gas mixtures illustrates the value of a sensitive and reliable oxygen monitor to provide a warning if there is any degradation in the product gas. Also, it shows the utility of an automatic backup oxygen supply incorporated into the breathing system that ensures delivery of 93% oxygen or greater following a decompression.
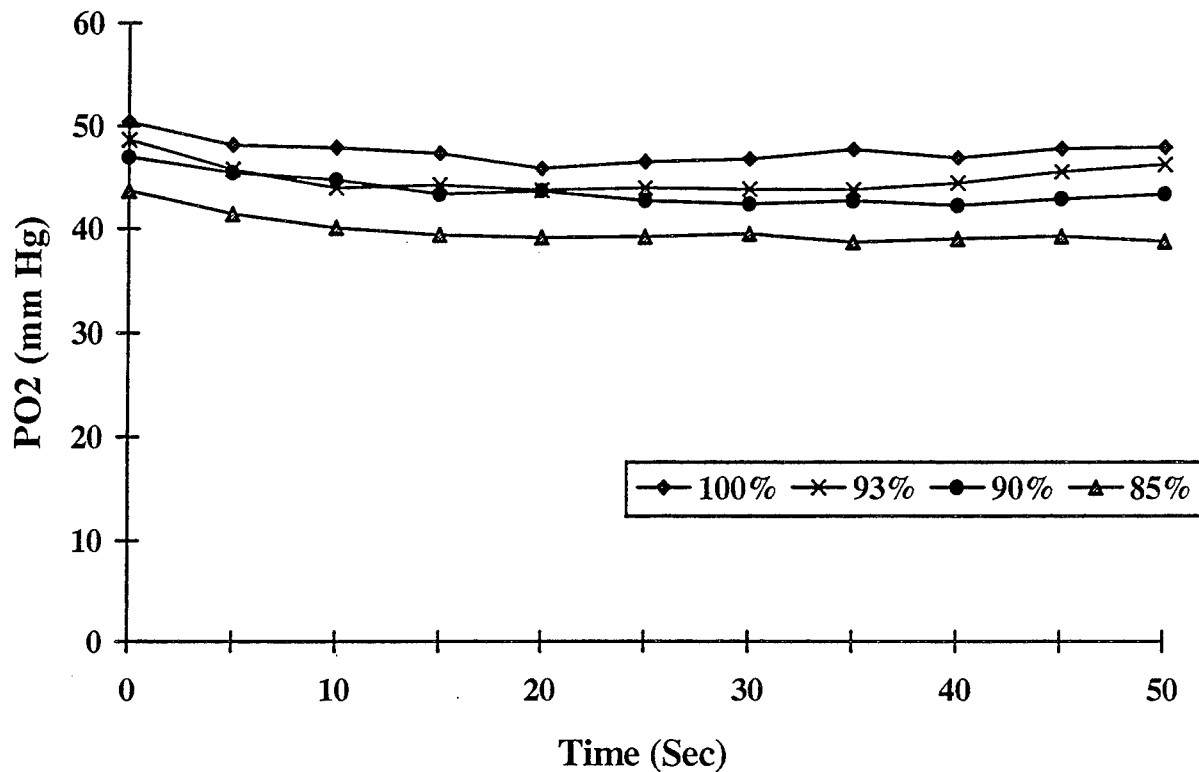
Figure 3. Mask Cavity Gas Concentrations vs. Time, Nondilution.

Figure 4 shows the end-tidal partial pressures of oxygen for the four breathing gases, before and after the RD, during the trials with the regulator in the dilution setting prior to the decompression. Once the RD occurred, the dilution port closed and undiluted breathing gas gradually replaced the oxygen/nitrogen mixture. The breathing regulator delivered between 40 and 50 percent oxygen when placed in the dilution mode. Thus, the partial pressures prior to the RD were considerably lower compared to the non-dilution trials. With no dilution, all of the oxygen partial pressures were 225 mm Hg or greater. For the dilution experiments, they fell to around 120 mm Hg or less.

Figure 5 shows the time frame during and after the RD for the dilution experiments. The most striking aspect is that, following the decompression, all four gas mixtures have led to partial pressures below the recommended 30 mm Hg limit. The 90% and 85% values remain below 30 mm Hg at the first 5 second interval.

The average oxygen partial pressures for each of the gas mixtures during the time at 50,000 feet were as follows: 100% oxygen - 42 mm Hg; 93% oxygen - 39 mm Hg; 90% oxygen - 37 mm Hg; 85% oxygen - 37 mm Hg. The values for 93%, 90%, and 85% were significantly different from the 100% results but not from each other. Again, this regulator did not produce the largest amount of dilution allowed by the specification. Had it done so, the partial pressures would have fallen even lower and been slower to rise.

Figure 6 makes it easier to see the impact of dilution. Here the oxygen partial pressures over time are shown for the 90% oxygen trials in both the non-dilution and dilution regulator settings. A large difference can be seen immediately following the RD and more than 20 seconds elapse before the end-tidal values are fairly equivalent.

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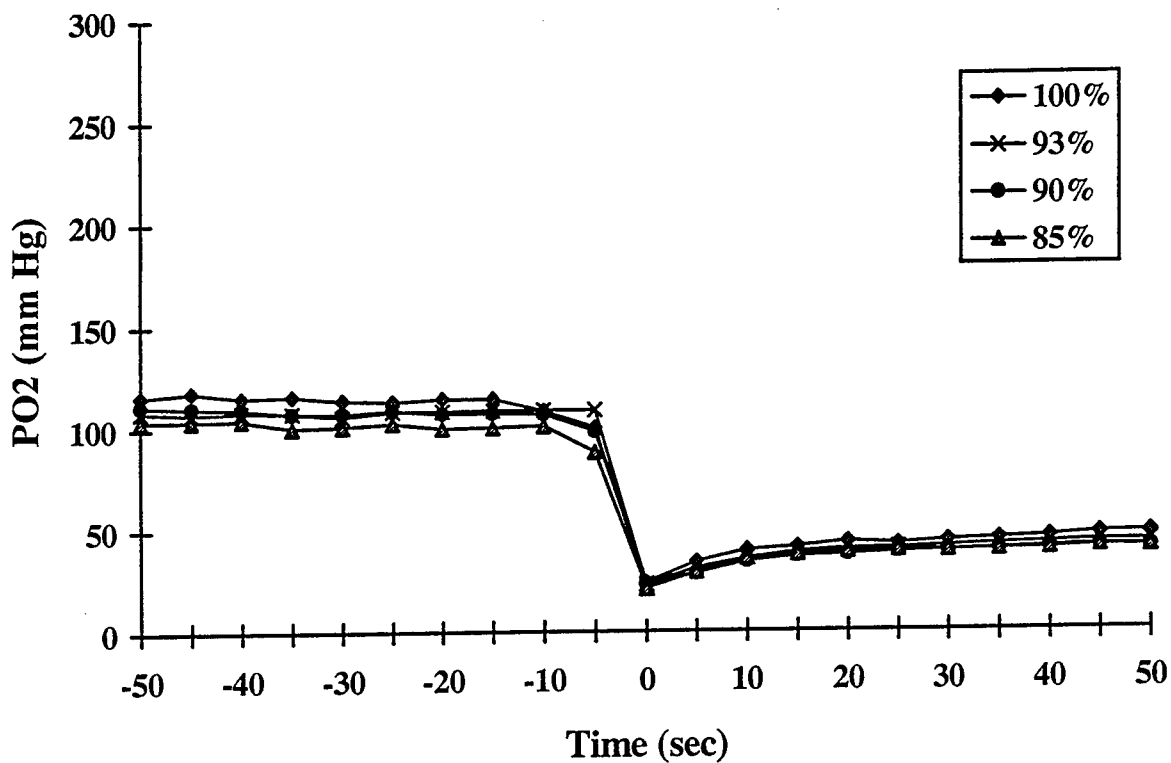
Figure 4. Mask Cavity Gas Concentrations vs. Time, Dilution.
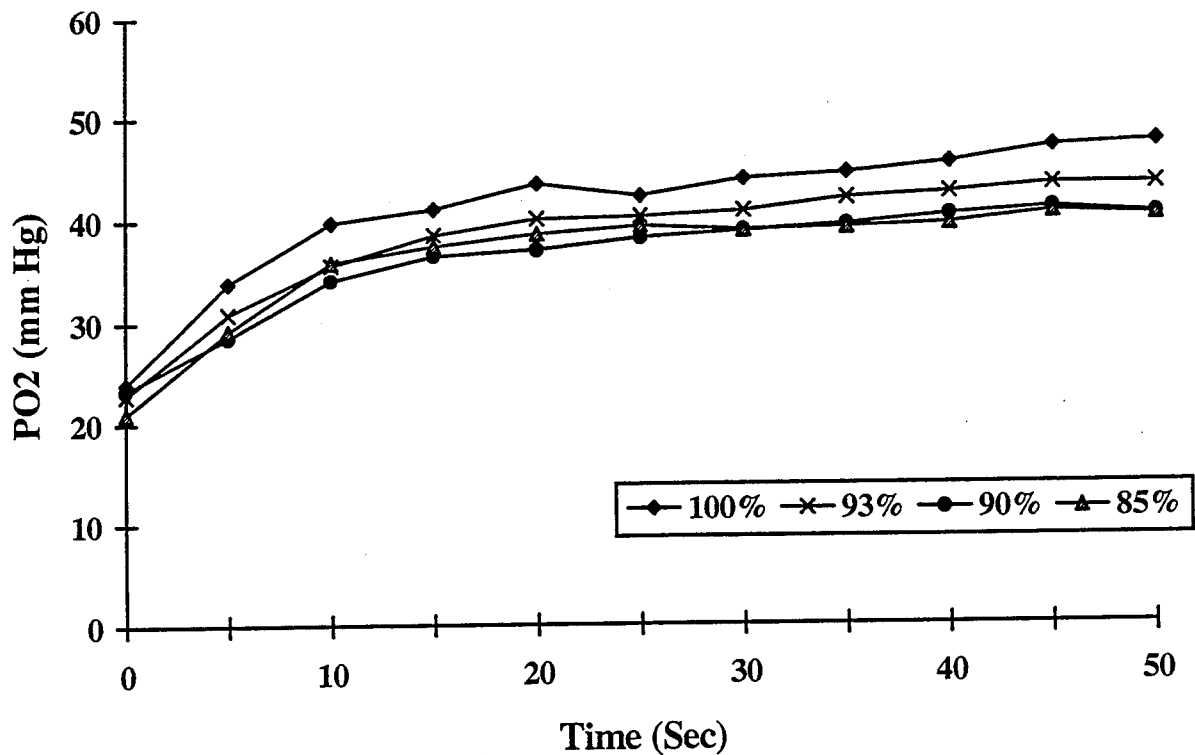
Figure 5. Mask Cavity Gas Concentrations vs. Time, Dilution.

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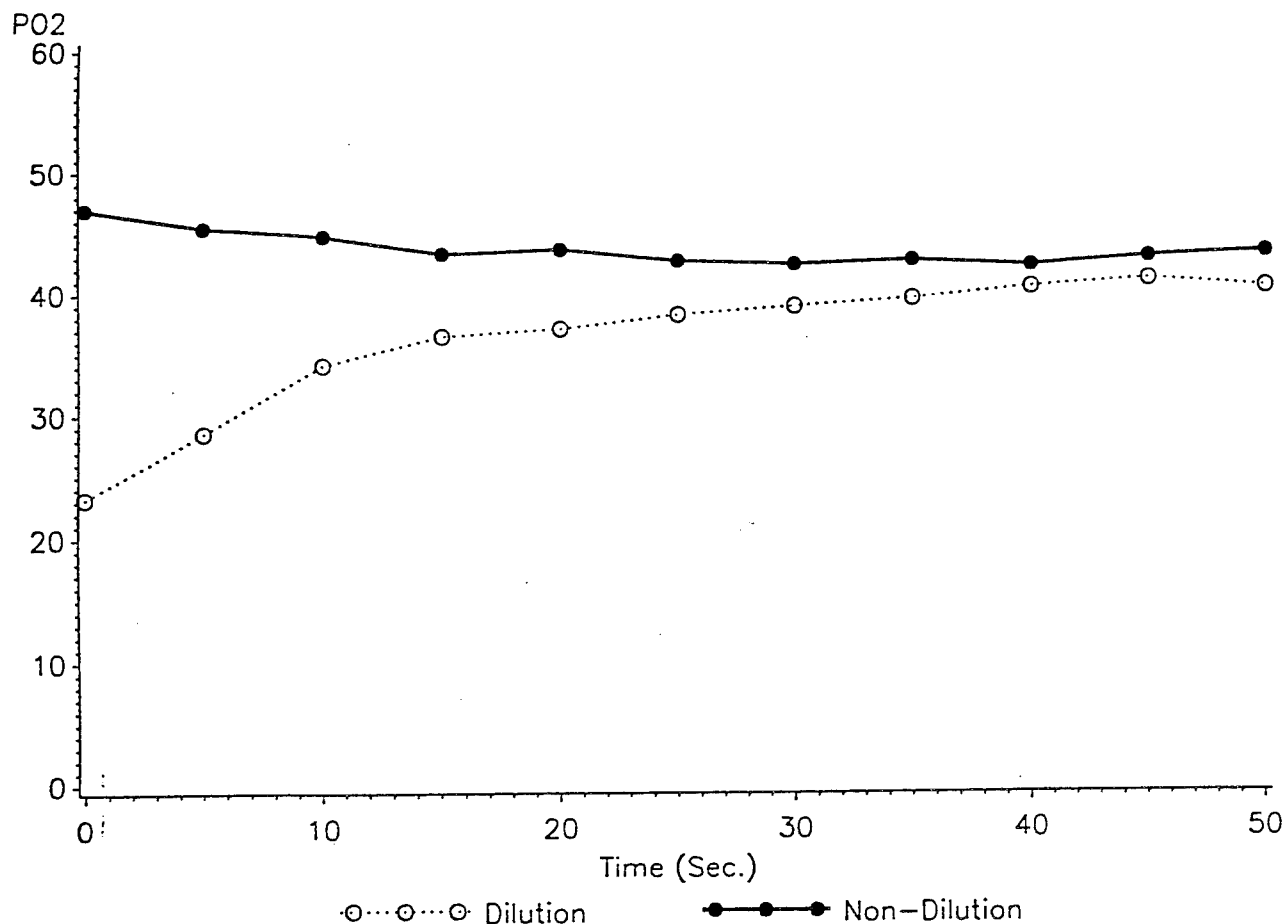
Figure 6. Mask Cavity Gas Concentrations for 90% O2 Mixture, Non-Dilution vs. Dilution.

Conclusions

The subjects in this study showed that it was possible to complete a one-minute exposure to 50,000 feet after a rapid decompression using the standard USAF breathing system. However, the hypoxia and performance impairment associated with 30 mm Hg pressure breathing at 50,000 feet was significantly worse than baseline measures, even for the 100% oxygen trials. It was concluded that the standard breathing system provided only marginal hypoxia protection at high altitude, regardless of the breathing gas.

One of the problems was the inability of the MBU-12/P mask to provide an adequate seal at 30 mm Hg of breathing pressure. Of the 17 subjects, 13 had to stabilize the mask by hand to prevent high (>30 L [ATP] /min) gas flow from the mask. This was in spite of the fact that great care was taken prior to each exposure to fit the subject's mask as well as possible and to test it for leaks. Normally, the area around the bridge of the nose failed to seal and allowed gas to blow into the eyes. Without stabilizing the mask, most of the subjects would have had difficulty performing the tracking aspect of the performance task. Not only does a leak from the mask frequently affect vision, it can also decrease the delivery pressure of the breathing gas. Fortunately, the CRU-73/A regulator was capable of delivering gas volume sufficient to overcome most mask leakage without a serious decrease in outlet pressure

As seen earlier, when the regulator was in the dilution mode, all of the breathing gases tested, including 100% oxygen, produced post decompression oxygen partial pressures below 30 mm Hg. Also, as mentioned earlier, those partial pressures would have dropped even lower if the regulator had performed at the low end of the specification for pressure breathing or had diluted the breathing gas to the level allowed by specification.

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In view of those problems, it was recommended that the emergency flight ceiling of 50,000 feet not be considered as the true routine use ceiling for the standard oxygen system, even with 100% oxygen as the breathing gas. Instead, it was recommended the system be employed strictly as a get-me-down system from 47,000 feet. For extended flight above 47,000 feet, the breathing system should have the following characteristics: 1) an increased positive pressure breathing schedule, on the order of 45-50 mm Hg at 50,000 feet; 2) an oxygen mask capable of sealing under breathing pressures of 50-70 mm Hg; and 3) inclusion of chest and lower body counterpressure.

Additionally, even though an analysis of the performance data found no significant differences for the tracking task between the 100% and 93% values, because the hypoxia impairment would be greater with 93%, it was suggested that prudent ceiling limits for that breathing gas would be 46,000 feet as the routine use ceiling and 48,000 feet as the emergency ceiling. However, because most of the problem with substitution of 93% oxygen for ABO was related to deficiencies in the standard breathing system, the following guidelines were supplied for use of OBOGS gas at the current emergency ceiling: EONS/OBOGS normal gas concentrations (93%-95% oxygen) can be breathed during emergency descent from 50,000 feet provided time at 50,000 feet is < 1 minute, time above 40,000 feet is < 3 minutes, time above 30,000 feet is < 5 minutes, mask fit is adequate, breathing pressure is adequate, and the breathing gas is undiluted by the regulator.

For OBOGS systems fielded on current aircraft, those recommendations have only been partially implemented. For example, for current flight tests of an F-16 OBOGS, the aircrew are wearing the Combined Advanced Technology Enhanced Design G Ensemble (COMBAT EDGE) and the aircraft has a CRU-98/A regulator. With COMBAT EDGE, the MBU-12/P has been replaced with the MBU-20/P mask that provides a better seal at high positive pressure breathing levels. Also, the HGU-55/P helmet has been modified with the addition of a bladder in the back to help improve the mask seal during PPB. A chest counterpressure vest has been added to the flight clothing ensemble to protect against over expansion of the lungs during high PPB. Finally, the CRU-98/A regulator, in comparison to the CRU-73/A, provides a more enriched oxygen concentration at high cabin altitudes to help prevent the drop in oxygen partial pressure during a rapid decompression. However, the pressure breathing schedule of the CRU-98/A is still the same as the CRU-73/A, that is, 30 mm Hg at 50,000 feet. Also, the system does not include an automatic backup oxygen system of 93% oxygen or greater.

The value of the suggested improvements to the standard breathing system was shown in a follow-on study to EONS called EONS II. For that study, the breathing gas was limited to 100% and 94% oxygen, again in both dilution and non-dilution regulator settings. Performance measurements were tested as in the original study. Altitude exposures consisted of one-second rapid decompressions (5 psid) to 46000, 52000, 56000, and 60000 feet. As with the original EONS study, time at peak altitude was 1 minute. The subjects wore the Tactical Life Support System or TLSS. In contrast to the standard oxygen system, TLSS provided: 1) a mask capable of sealing at high breathing pressure; 2) helmet assisted mask tensioning; 3) chest counter pressure equal to breathing pressure; 4) an extended coverage G-suit; 5) and a regulator capable of delivering breathing pressures for altitude up to 70 mm Hg. Breathing pressures for EONS II were 50 mm Hg at 46,000 feet and 70 mm Hg at each of the other altitudes. Also, in contrast to the first EONS study, EONS II actually used inflation of the G-suit at peak altitude during positive pressure breathing. The G-suit to breathing pressure ratio was 3.5:1. At least 13 subject exposures were completed at each altitude.

With the improvements to the breathing system that TLSS provided, the physiological measurements during RDs to 60,000 feet using TLSS and 93% oxygen were better than those seen in the original EONS study at 50,000 feet using 100% oxygen and the standard oxygen system. Additionally, subjects who had participated in both EONS studies reported that, when wearing TLSS during a 60,000 feet decompression, the level of protection subjectively felt much better than using the standard oxygen system at 50,000 feet.

Overall tracking task performance results for EONS II showed no differences between the OBOGS breathing oxygen concentration of 94% and ABO. However, significant RMS error differences were found between the ground level and base altitude trials compared to peak altitude trials. The high positive breathing pressures occurring at the peak altitudes was apparently the difference.

Two last points on the EONS studies dealt with PPB syncope and decompression sickness. One of the most striking findings of the original EONS research was the effect of pressure breathing experience on the incidence of syncope in the subject population. During the experiments there were 4 incidents of loss of consciousness (LOC) within one minute after the decompression (1 non-dilution trial, 3 dilution). Most of those incidents occurred early in the study when 100% oxygen was the breathing gas. Later exposures employed breathing gas mixtures with significantly lower oxygen concentrations. However, in spite of increasing levels of hypoxia, the incidence of pressure breathing syncope declined. The decline was attributed to a training effect due to increased exposure to 30 mm Hg PPB. Active duty aircrew receive refresher physiological training once every three years. It's possible that such infrequent training may not sufficiently prepare a crewmember to cope with positive pressure breathing at 30 mm Hg. There were no incidents of LOC during the EONS II study which involved 82 manned exposures that all used higher levels of PPB than the original EONS study. Two explanations are possible. First, subjects in the EONS project did not have the benefit of any inflation of the G-suit during PPB. As mentioned earlier, EONS II employed a G-suit to breathing pressure ratio of 3.5:1. Second, a more intensive ground level training program on PPB was instituted for EONS II.

Finally, with regard to decompression sickness, neither EONS study had any reported incidents of DCS, despite the fact that on 47 of the experimental trials in EONS II, the chamber altitude profile was modified to include a 10-minute period at 40,000 feet following the one minute stay at peak altitude. That modification was designed to help investigate the DCS risk following a high-altitude exposure. The lack of DCS problems may be explained by the 60 minutes of oxygen prebreathe used in each study and the still relatively short exposure to altitude.

Discussion

DR. ACKLES: Did you say at the very beginning of the paper that you used an oximeter?

CAPT. O'CONNOR: Yes.

DR. ACKLES: You didn't report any saturation values. What were your saturations at the lower levels?

CAPT. O'CONNOR: Around 80% was the lowest we saw, but we didn't have the greatest confidence in the oximeter data. We were unable to wear it on the hand used for the performance tracking task and needed to stabilize the mask with the other hand during PPB. We got a fair amount of cutting in and out of the recordings.

MAJ. MILLER: On the EONS II study, when you were using 100% and 93%, how large was your subject pool and what was their experience level?

CAPT. O'CONNOR: Some of the subjects were in the original EONS study so their experience was very high. I'd have to go back and look to see exactly what the numbers were. A guess would be 15 subjects, but I'd have to look at it again.

COL. SHAFFSTALL: Did you document subjective hypoxia symptoms during the study for the high altitude portion?

CAPT. O'CONNOR: Yes, if I remember correctly, when they were analyzed there wasn't any difference between the 100% and 93%. It wasn't analyzed for 90% and 85%.

DR. BOMAR: I have a comment related to that. We routinely saw PCO2s in the 20 mm Hg range and some even down in high teens. A lot of the reported symptoms probably would be more attributable to these low PCO2's than hypoxia. Regardless of how many of the runs each of us completed, we all hyperventilated while we were pressure breathing and it became worse as the mask pressures were increased. The impact of training on the operational crews, if we move to a system like this, is going to be important. How many people currently check their mask for pressure breathing before they go fly?

MAJ. NEUBECK: All fighter aircrew are required to check their masks before each flight.

DR. BOMAR: At what pressure?

MAJ. NEUBECK: I don't know what pressure the regulator provides.

CAPT. O'CONNOR: We took a lot of care before each trial that each subject was fitted properly and still, as I mentioned, almost all of them had massive gas leaks using the MBU-12/P mask and had to use the one hand to stabilize the mask.

PROF. ERNSTING: Do you remember what pressure the regulator delivered at 50,000 feet?

CAPT. O'CONNOR: Thirty mm Hg.

PROF. ERNSTING: Was it right on 30 mm Hg?

CAPT. O'CONNOR: Yes.

PROF. ERNSTING: We conducted a whole series of rather similar studies in the 1960s and we found that it was absolutely critical to maintain 30 mm Hg mask pressures. Our equipment manufacturer was extremely upset because I had originally suggested the delivery range should be 30 to 32 mm Hg mean mask pressure and they

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asked us whether they could widen that up to 4 mm Hg. In our study, we found that if we went any lower than 30 mm Hg then we got disturbances of consciousness. If we went above 34 mm Hg, we saw more faints, so I fully support Dr. Bomar's comments about the low PCO2s. I certainly think the output pressure range the manufacturer allowed in his specifications on the old panel mounted regulators is absurd. The British Oxygen Company was very unhappy when we established our limits. We know in the UK, at least, that those limits are kept very tightly, provided the mask doesn't leak.

CAPT. O'CONNOR: I should have also mentioned that, by implementation of the COMBAT EDGE system in at least the F-16 now, some of our problems have been reduced. We now have a better mask in the MBU-20/P. Also, in the F-15E, the CRU-98 regulator has a more enriched dilution schedule at the high cabin altitudes. However, we still have many of the older regulators in the inventory that provide a much wider range of breathing pressures at altitude.

DR. BOMAR: You selected a regulator that was at the high end of the pressure output spectrum.

CAPT. O'CONNOR: Exactly.

DR. SEARS: In a review of the MILSPEC for oxygen systems, we found that 20 mm Hg was listed as the low end of the range for output pressure at 50,000 feet. This really is too low to sustain consciousness.

CAPT. O'CONNOR: And that doesn't address potential mask leaks.

DR. SEARS: Right. It doesn't address the mask seal leaks.

DR. BOMAR: The MILSPEC also does not establish oxygen pressure breathing requirements above 47,000 feet.

Evolution of High-Altitude Protection and Operational Aircrew Training in the RAF

A J F Macmillan RAF, BSC, MB, ChB, MFOM

Introduction

In the years that followed the end of World War II, development of bomber and interceptor aircraft capable of operating at high altitude made rapid progress. Although these aircraft had pressurization systems that limited the maximum cabin altitude to 25,000 feet or thereabouts, the risk of failure of the pressurization system due either to mechanical fault or enemy action was considered to be high. The principal physiological hazards associated with loss of cabin pressure at altitudes greater than 30,000 feet are hypoxia, decompression sickness and hypothermia. A full-pressure suit assembly is necessary if protection is required against all three hazards over a prolonged period. However, if the aircraft can descend promptly and rapidly (within 3-4 minutes) to an altitude of less than 40,000 feet, protection will be required only against hypoxia. A full-pressure suit assembly will provide the ideal physiological protection but it is bulky, cumbersome, impairs operational efficiency during routine flying, and, with an intact cabin, imposes major ground procedural problems, particularly when rapid response is required. When a "get-you-down" facility only is acceptable, the hypoxia induced by exposure to high altitude can be prevented by pressure breathing and application of counterpressure to certain areas of the surface of the body to limit the respiratory and cardiovascular disturbances resulting from raised intrathoracic pressure. The major advantages of a partial-pressure assembly include less restriction when the garments are inflated or uninflated, thus providing greater routine comfort and a lower thermal load. To exploit fully these advantages it is highly desirable that counterpressure should be applied to as small an area of the body as possible. Thus, the design of partial-pressure assemblies represents a compromise between ideal physiological requirements and functional convenience. The technique of pressure breathing with 100% oxygen and the application of limited counterpressure to the body was adopted by the Royal Air Force in 1954 as a method of providing short duration protection against hypoxia at altitudes above 50,000 feet.

Breathing 100% oxygen at 40,000 feet (alveolar PO_2 55-60 mm Hg) causes only mild impairment of performance, whilst the impairment produced by breathing 100% at 44,000 feet (alveolar PO_2 40 mm Hg) demands that the partial pressure in the lungs must be maintained greater than the absolute pressure at this altitude. Thus, although the range of PO_2 within the lungs may be acceptable over a short period of time between 115 and 140 mm Hg absolute, the level that is required is a compromise between that needed to prevent significant hypoxia whilst minimising the respiratory and cardiovascular effects of raised intrapulmonary pressure. Thus, in systems in which the physiological disturbances induced by raised intrathoracic pressure can be minimised by extensive coverage of the body surface with counterpressure garments, the acceptable level of hypoxia may be greater than in those systems where coverage is minimal.

The development of partial-pressure assemblies within the Royal Air Force evolved, therefore, through examination of the acceptable compromises between raised intrathoracic pressure and the effects this had on the head and neck, respiration, and circulation. The criteria for acceptance of a partial-pressure assembly are shown in Table 1.

Table 1

Criteria for Acceptable Partial-Pressure Assemblies
- No Impairment of Performance
- No Excessive Hypocapnia
- Heart rate less than 130/min
- No Impending Vasovagal Syncope

Effects of Pressure Breathing on the Head and Neck

In the presence of effective thoracic and abdominal counterpressure, the factors limiting the level of pressure breathing that may be delivered to the respiratory tract are the undesirable effects on the head and neck. The most convenient method of delivering breathing gas to the respiratory tract is via an oronasal mask capable of sealing the necessary mask cavity pressures. Typical RAF oxygen masks (P/Q), when properly fitted and adjusted, will seal mask cavity pressures up to 100 mm Hg without significant leakage. In practice, however, because support is given to a limited area of the face, well-defined physiological effects limit the pressure that can be tolerated. Distension of the upper respiratory tract becomes very uncomfortable at pressures in excess of 70 mm Hg and this discomfort is the main limitation to use of an oronasal mask. Additional effects such as blepharospasm (due to gas passing up the naso-lachrymal ducts) and rupture of conjunctival vessels, as a consequence of raised intrathoracic (and hence arterial) blood pressure, limit breathing pressure and duration for which an oronasal mask may be used at 70 mm Hg to 3-4 minutes. However, well-trained subjects can tolerate pressure breathing up to 80 mm Hg for short periods and, provided an aircraft can initiate descent within 1 minute of the onset of loss of cabin pressurization and continue descent at a rate in excess of 10,000 feet per minute, mask cavity pressures of up to 80 mm Hg, accompanied by adequate body counterpressure should be acceptable. A full-pressure or partial-pressure helmet would eliminate problems associated with the raised upper respiratory tract pressure; however, these helmets are bulky, heavy, cumbersome and probably inappropriate for use in the next generation of high-performance aircraft.

Effects of Pressure Breathing on Respiratory System

Positive pressure breathing produces lung distension and individuals have to make a constant expiratory effort to minimize this distension. Consequently, hyperventilation and respiratory muscle fatigue are common accompaniments of the procedure. Whilst a counterpressure waistcoat applied to the chest provides some support, unless the remainder of the trunk is also supported in a similar fashion, fatigue of the abdominal muscles will occur. The most effective method of applying such counterpressure (other than by means of gas as is done in a full-pressure suit) is to encircle the trunk with a gas bladder covered by an external restraining layer so that when it is inflated with gas, the pressure of the bladder is exerted on the surface of the trunk through the inner layer of the bladder and any clothing worn beneath the garment. This principle was utilised in the development of the Royal Air Force pressure jerkin system, the bladder of the pressure jerkin is connected to the breathing regulator so that the pressure within the bladder always equals that delivered to the respiratory tract. The garment was developed as the outermost layer of the aircrew equipment assembly (AEA), and so minimised the encumbrance on the ground and reduced the heat load, since it was the last garment to be donned prior to flight and the first removed. Disadvantages, however, accrued since survival aids and flotation devices were required to be integrated as part of the assembly.

Pressure Breathing Effects on Cardiovascular System

The effect of raised intrathoracic pressure produced by positive pressure breathing is displacement of blood into the peripheral circulation, especially in the limbs. The effective blood volume was reduced by this initial pooling of blood in the limbs and, subsequently, by passage of fluid from the blood into the extra vascular space due to the raised transmural pressures of the capillaries. Vasovagal syncope may occur when the total reduction of total blood volume exceeds about 700 ml. Other effects of pressure breathing (discomfort, pain, hyperventilation, and hypoxia) reduce the decrease in effective blood volume required to induce syncope during pressure breathing. Reduction of effective blood volume may be prevented by applying counterpressure to the limbs. Thus in the early partial-pressure assemblies, Royal Air Force equipment included the standard anti-G suit that applies counterpressure to the skin of the lower limbs. In the systems developed for bomber aircraft, the anti-G trousers were also connected to the breathing gas supply; but in aircraft in which the G trousers were already worn by aircrew for protection against acceleration, the pressure in the anti G trousers in some systems was raised to 40 mm Hg greater than that applied to the respiratory tract, so that the blood content of most of the lower limbs is reduced during pressure breathing below that which existed at rest. When breathing pressure is high, or hypoxia is increased, the reduction of effective blood volume may be decreased still further by applying counterpressure to the upper as well as the lower limbs. extending the bladder coverage to the upper limbs reduces comfort and mobility and increases heat load. Thus, counterpressure to the upper limbs has been avoided whenever possible.

UK Partial-Pressure Assemblies

Pressure breathing mask alone. The RAF type P/Q series of pressure breathing masks used with the pressure demand regulator capable of delivering positive pressure of 30 mm Hg at 50,000 feet will provide protection for one minute at 50,000 ft if followed by descent at 10,000 ft per minute to below 40,000 feet.

Pressure breathing mask and pressure jerkin. An assembly consisting of the type P/Q mask, pressure jerkin and a delivery system providing 56-58 mm Hg at 52,000 ft will provide protection for one minute at 52,000 feet if followed by descent at 10,000 feet per minute to below 40,000 feet.

Pressure breathing mask, pressure jerkin and anti-G suit. This was the most widely utilised system within the Royal Air Force for protection against hypoxia at cabin altitudes up to 56,000 feet. The positive pressure breathing required from the demand regulator is 70 mm Hg at 56,000 feet. The limited protection provided by this assembly is 30 seconds at 56,000 feet followed by descent at 10,000 feet per minute to below 40,000 feet.

Partial-pressure helmet, pressure jerkin and anti G suit. A form of partial-pressure helmet was developed for those aircraft in which protection at altitudes greater than 56,000 feet was required. The panel-mounted pressure demand regulator used with this assembly raises the helmet pressure to 110 mm Hg at 66,000 feet. The limited protection provided by this assembly is 1 minute at 66,000 feet followed by descent to below 40,000 feet at 10,000 feet per minute.

Partial-pressure helmet, sleeved pressure jerkin and anti G suit. Experimental investigation of this type of assembly was conducted in the late 1950s to provide protection at cabin altitudes up to 100,000 feet. The breathing pressure required at this altitude was 140 mm Hg, and whilst experimentally, protection could be provided for 1 minute at 100,000 feet followed by descent to below 40,000 feet at 15,000 feet per minute, ebullism producing gross swelling of the hands at altitudes greater than 65,000 feet was invariably present.

Pressure breathing mask and combined partial-pressure suit. In the early 1960s, a partial-pressure garment that incorporated ventilating air and with the pressure breathing counterpressure bladder covering the trunk and lower and upper limbs was developed. A separate set of bladders over the abdomen and lower limbs was separately connected to the anti-G valve, thus, this double layer of bladders around the lower body permitted adequate counterpressure to be provided even after separation from the anti-G system. An assembly of this nature was developed for the Phantom

interceptor aircraft and with a man-mounted miniature regulator delivering a maximum of 70 mm Hg, protection was provided for one minute at 60,000 feet followed by descent at 10,000 feet per minute to below 40,000 feet.

Partial-pressure helmet and combined partial-pressure suit. An assembly comprised of a partial-pressure helmet and a combined partial-pressure suit inflated by a torso mounted miniature regulator capable of maintaining an absolute pressure of 145 mm Hg in the helmet was used to prevent hypoxia at cabin altitudes up to 75,000 feet. An assembly of this nature was utilised in the early test flying phase of the supersonic transport aircraft Concorde. The assembly was part of the integrated escape system for the test aircrew who would have been required to exit the aircraft through explosive hatches in the event of serious malfunction at high altitude.

Training and Use of Partial-Pressure Assemblies

Since the mid-1970s, the high-altitude role of the RAF has been reduced essentially to one aircraft type. Apart from this (a reconnaissance role), the expected operations were considered to be at low level. Recent experience of training in the use of partial-pressure assemblies has therefore been very limited. Over the years, however, well over 1,000 Royal Air Force and Royal Navy aircrew have been trained in the use of one or more of the 3 commonly used partial-pressure assemblies in the UK. A typical course includes instruction in the physiology of pressure breathing, and mode of operation of the assembly and its supply system. The oronasal mask and pressure clothing are fitted and then each individual is given training in pressure breathing at ground level. The magnitude of the pressure and the duration of exposure are increased from one training session to the next until the breathing pressure/time profile to which the trainee will be exposed in the decompression chamber is reached (Tables 2,3,4). Following successful completion of the ground level training, each individual, after a minimum of 30 minutes pre-oxygenation, is exposed in a decompression chamber to an altitude profile appropriate to the assembly. Heart rate is monitored during exposure to high altitude.

Table 2

Mask pressure jerkin and anti G suit assembly - training schedule

Ground level pressure breathing

      30 mm Hg for 2 minutes, on one occasion
      45 mm Hg for 3 minutes, on one occasion
      60 mm Hg for 1½ minutes, on two occasions
      70 mm Hg for ½ minute, decaying to 0 mm Hg over the subsequent 1½ minutes, on one occasion

Decompression chamber exposure

      Rapid decompression (3-5 seconds) from 25,000 feet to 56,000 feet
      Maintain 56,000 feet for 30 seconds
      Descend to 38,000 feet at 10,000 feet per minute

Table 3

Mask and combined partial-pressure suit assembly - training schedule

Ground level pressure breathing

        30 mm Hg for 2 minutes, on one occasion
        45 mm Hg for 3 minutes, on one occasion
        60 mm Hg for 1½ minutes, on two occasions
        70 mm Hg for 1 minute, decaying to 0 mm Hg over the subsequent 2 minutes, on one occasion

Decompression chamber exposure

        Rapid decompression (3-5 seconds) from 25,000 feet to 60,000 feet
        Maintain 60,000 feet for 1 minute
        Descend to 38,000 feet at 10,000 feet per minute

Table 4

Pressure helmet, pressure jerkin and anti G suit assembly - training schedule

Ground level pressure breathing

        45 mm Hg for 4 minutes, on two occasions
        80 mm Hg for 4 minutes, on two occasions
        100 mm Hg for 2 minutes, on two occasions
        100 mm Hg for 1 minute, decaying to 0 mm Hg over the subsequent 3 minutes, on one occasion

Decompression chamber exposure

        Rapid decompression (3-5 seconds) from 25,000 feet to 60,000 feet
        Maintain 60,000 feet for 1 minute
        Descend to 38,000 feet at 10,000 feet per minute

In our experience of this form of pressure breathing training, only 5% of exposures are required to be terminated prematurely, the majority of these have been caused by severe gut pain or signs and symptoms of incipient vasovagal syncope. During the hypobaric exposures in UK utilising these profiles for aircrew training, no premature descents were caused by symptoms of decompression sickness.

The future of partial-pressure assemblies is seen as providing short duration protection against hypoxia following loss of cabin pressurization or ejection at high altitude. A very effective assembly that will provide protection at cabin altitudes probably up to 70,000 feet will consist of an oronasal mask, a lightweight pressure waistcoat, and anti-G suit. Detailed discussions of these assemblies are contained in later presentations.

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Discussion

DR. MOON: I would like to support your viewpoint on PCO_2. I think it's very important. In some of our studies we found that the degree to which the brain can survive, in hypoxia, is very strongly dependent upon PCO_2, and in fact the brain can tolerate much more hypoxia when the PCO_2 is higher than normal than when its normal or certainly below normal. So if the PCO_2 in some fashion could be allowed to rise, the requirements for PO_2 could possibly be relaxed. If the system could be designed, or by training, such that PCO_2 in fact were elevated, this would help prevent loss of consciousness due to the low PCO_2.

DR. MACMILLAN: Yes, I think that is a valid point, but again I must say you shouldn't just base the effectiveness of the system on these measures alone. From the operational point of view it's circulatory support as well as the maximum permissible degree of impairment of hypoxia, and they shouldn't be taken in isolation.

COL. SHAFFSTALL: Relative to the UK's experience and ours in training centrifuge and chamber subjects to use positive pressure breathing systems, the training for both environments is extremely critical for proper use of the equipment. It may also help with the PCO_2 problem at altitude. Have you been looking at the training requirements from any aspect?

MS. MCGARVEY: From the F-22 systems program office (SPO), I can tell you that the contractor is putting together a training syllabus and it does include the life-support training for high G as well as for altitude. The centrifuge training is in line with the COMBAT EDGE training that the ACC has now implemented.

COL. SHAFFSTALL: Has anyone in our aerospace physiological units been brought in on the training discussions?

COL. SHERMAN: Not that I know of at this time.

Raising the Ceiling to 80,000 ft.

A Historical Review of High-Altitude Research at DCIEM

Kenneth N. Ackles, Ph.D.

Introduction

I was first introduced to high-altitude physiology and positive pressure breathing (PPB) at the RAF Institute of Aviation Medicine by John Ernsting while completing a Post Doctoral Fellowship in Aviation Medicine in 1966/67. I had come straight out of university and knew nothing about altitude physiology. On my return to Canada, I immediately was placed in charge of the Diving Physiology program. However, the lessons learned at Farnborough were well learned because in 1975, when I was consulted on the requirements for altitude protection in Canada's proposed new fighter aircraft (NFA), I was able to mount a project that became known as Project Phoenix.

This Statement of Requirement stated that the NFA would be required to cruise at 60,000 ft. and pop-up for weapons delivery to 80,000 ft. Most fighters of the day had operational ceilings of 50,000 ft. The challenge was to meet the operational requirements without resorting to a full-pressure suit, which we did not consider compatible with fighter operations.

First of all, we completed a survey of the state of the art in altitude protection systems. The RCAF IAM had done much of the early work on PPB at high altitude, but reports were very hard to find. We then visited Brooks and Wright Patterson AFBs, and NADC Warminster to discuss the USAF and USN full-and partial-pressure suit experience, this was followed by a visit to RAF/IAM where we were brought up to date on the PPB jerkin and G-suit, but I found that nothing very much had changed over past the 10 years as our allies didn't have an operational requirement for such high altitudes at that time.

At this time we also became aware of the new PPB ensemble developed by the RSAF, comprised of a mini-abdominal bladder and 3.2 PPB/G-suit ratio (Larsson & Stromblad, 1967). The RSAF claimed in their paper that their much-reduced abdominal bladder combined with the increase in G-suit pressure provided similar protection to the RAF jerkin and G-suit ensemble with equal pressure in the jerkin and G-suit. We reasoned that if the Swedish suit with its much reduced jerkin could equal the protection of the RAF ensemble, then perhaps the RAF ensemble would provide greater protection if it was used with an increased PPB/G-suit pressure ratio.

Ground Level Experiments

To explore this theory, we planned a very ambitious experiment to compare three suits covering a total of 10 PPB conditions and 10 subjects. This resulted in a 10 X 10 Latin square design – 100 individual experiments that we completed in about 6 weeks. We selected PPB levels of 50 and 70 mm Hg and investigated jerkins from the RCAF, RAF, RSAF. The G-suits were very similar. we used PPB/G-Suit pressure ratios of 1 & 3.2, except for the Swedish suit for which we used only with a ratio of 3.2. The PPB ratio of 3.2 was what had been used by the RSAF (Larsson & Stromblad, 1967) . The results of these experiments have been reported (Ackles, et al., 1978) .

In summary, we eliminated the early RCAF jerkin from further consideration as it was quite inadequate at the higher PPB pressures. We confirmed the Swedish claim that RSAF suit with a 3.2 ratio gave equal protection to the RAF ensemble with a ratio of one. However, as we had speculated, the RAF ensemble with ratio of 3.2 gave the greatest protection against physiological effects of PPB. In this experiment we began a long study of the cardiovascular effects of PPB using impedance cardiography. Although at the time we were mildly criticized for using a technique that did not have a direct calibration, we were convinced that we were at least accurately

measuring relative changes in stroke volume and cardiac output. I believe that subsequent publications have validated our initial convictions.

The next series of experiments looked at PPB G-suit ratios of 1, 2, and 3.2 (using RAF ensemble). It was confirmed that of these ratios, 3.2 gave the best protection. We always defined the best protection as that ensemble or ratio that resulted in the least change in cardiovascular parameters from control during the period of PPB. Next we examined a ratio of 4 that proved to be better that 3.2. Ratios higher than 4 were not evaluated, as by this time the G-suit was getting quite uncomfortable during the ground-level PPB and the G-suit pressure at 280 mm Hg was higher than the arterial pressure.

High Altitude Experiments

The next series of experiments looked at PPB at altitude. We felt that we had established an optimal protection package with the RAF jerkin and G-Suit and a PPB to G-Suit ratio of 4. Since DCIEM had not done any high-altitude experiments for several years, we started by following a training program based on an RAF IAM report. Starting with explosive (< 0.3 seconds) decompressions to 56,000 ft for 3 minutes with 70 mm Hg, we soon progressed to 60,000 ft with the same 70 mm Hg PPB. As expected, the RAF jerkin and G-suit gave excellent protection with a ratio of 4. At this time, we also realized the limitation of high levels of PPB with using only a mask and not a pressure or partial-pressure helmet. Some preliminary tests also showed that the RAF P/Q masks would not seal reliably beyond 80 mm Hg. With the maximum level of PPB established at 80 mm Hg, the question then was if our target altitude of 80,000 ft would be attainable.

In all these high altitude experiments, we prebreathed 100% O_2 for at least one hour prior to going to altitude. However, with all the control measurements and altitude excursions for gas release, the subjects were on O_2 for at least 90 minutes, and most closer to 120 minutes. We never had any problem with decompression sickness, but our exposures were relatively short. Descents from altitude were at a rate of 20,000 ft/min.

The TTCP WP 61 Air Standard on High Altitude stated at that time that the inspired pO_2 must be at least 120 mm Hg (hence 70 mm Hg PPB at 60,000 ft. (56 mm Hg) was OK). It was decided to progress carefully with complete studies at 60,000, 72,000, and if possible, 80,000 ft (72.000 rather that 70,000 since it was about halfway between 60 and 80,000 ft with respect to pressure).

The real aim of this series of experiments was to determine what degree of hypoxia was still compatible with full consciousness. The first series was 60,000 ft for 3 minutes with 60 mm Hg PPB (PI_{O2} = 114 mm Hg), which was uneventful. The next series to 72,000 ft for 2 minutes with 80 mm Hg (PI_{O2} = 110 mm Hg) was again uneventful, although here we were naturally concerned about exposure to less that 47 mm Hg because of tales of body fluids boiling away. Remember, this was in 1977 and our state of knowledge of such things was not as advanced as it is today. Again the series was completed with no problems and so we repeated the 72,000 ft exposures but this time with a PPB of 70 mm Hg (PI_{O2} = 100 mm Hg). Our principal psychomotor task was the RAF IAM mannikin test. Again, for the length of the exposures, the series was successful. We were especially watching the arterial oxygen saturation during all these exposures and had developed a rule of thumb that we would not allow the saturation to drop below 65% at any time. Following this success and having equaled the PI_{O2} at 80,000 ft (100 mm Hg), we cautiously did the rapid decompression series to 80,000 for 1 minute with a PPB level of 80 mm Hg (PI_{O2} = 100 mm Hg). Here again,, our main concern was ebullism and not hypoxia. Again, the series was completed successfully without incident. I believe that I reported the only interesting phenomena due to the extremely low atmospheric pressure (20 mm Hg). With the 80 mm Hg of PPB, there was considerable tearing in the eyes, so much so that it was at times difficult to see the mannikin test figures. I noticed that my eyes became quite cold (not uncomfortably so) due to the boiling away of the tears due to the low atmospheric pressure.

From these quite extensive physiological and the rather limited performance results, we postulated that emergency get-me-down escape was probably possible from altitudes as high as 80,000 ft. We had shown that a human could survive the hypoxia and high levels of PPB at altitudes as high as 80,000 ft with PPB as high as 80

mm Hg. The question remained whether a pilot could successfully recover a high-performance aircraft under these conditions.

When the results of these experiments were presented to WP 61, two Air Standards were rewritten to take our results into account. The minimum allowable PI_{O_2} was reduced to 100 mm Hg (ASCC WP 61, 1988) and the maximum PPB pressure a mask was to hold was increased to 82.5 mm Hg (ASCC WP 61, 1982).

Flight Simulator Experiments

To answer the question "could the pilot fly the aircraft" an interesting series of experiments was devised using a simple non-motion base T-33 flight simulator which was available in our laboratory. We developed a flight profile starting with straight and level flight at an altitude of 40,000 ft with a heading of 360°. At the signal of a decompression, the test subject was to immediately throttle back to idle, deploy the speed brakes and start a single spiral decent to the left or right to end up at 20,000 ft again with a heading of 360° while maintaining complete control of the aircraft. Each descent was scored on root mean square deviation from the ideal profile by recording outputs from the primary flight instruments. The subjects were trained fighter pilots and an equal number of technicians and scientists from DCIEM who were trained to fly the profiles, the reasoning being that pilots were highly trained so their responses might be considered to be almost reflexes, whereas the non-pilot subjects would perhaps be more sensitive to the experimental conditions. However, the pilots were required to confirm that we were running an experiment from which the results could be extrapolated to a true flight situation.

Following extensive training, we first investigated the effects of high levels of PPB on flying performance. No decrement in performance with PPB levels up to 80 mm Hg for the 2-3 minutes required to complete the profile was observed. The effects of hypoxia were then examined by providing hypoxic breathing mixtures that maintained the arterial saturation as close to 65% as possible. Again, no measurable effect on flying performance.

The final series of experiments combined the effects of PPB with hypoxia. In order to do this, we had to provide extremely hypoxic mixtures to the subjects who were pressure breathing at 80 mm Hg. Under this the most difficult condition, the non-pilots showed slightly more deviation from the ideal flight path than the pilots, but at no time did anyone lose control of the aircraft and all profiles were completed successfully. We interpreted the results of this series of experiments as an indication that there was a good possibility that our original aim of developing a protective system for emergency get-me-down from altitudes as high as 80,000 ft without the use of a full-pressure suit was possible.

Following these simulator experiments, we evaluated and tested a new jerkin design that had less coverage than the RAF jerkin and had a more pleasing appearance. While not having quite the protection of the RAF jerkin, the new Canadian jerkin, as it was known, did provide adequate protection as long as the G-suit:PPB ratio was 4:1. CF Jerkins of this design were also subsequently loaned to USAFSAM under a WP61 TPA for the early PPG experiments.

Flight Trials

As one final check, we conducted a flight trial in which we decompressed the cockpit during flight. This took place in 1979. A dual CF-104 was equipped with a special PPB regulator, and with the pilot wearing the new CF jerkin and standard G-suit with an RAF P/Q mask and the safety pilot fitted with a USAF full-pressure suit, we conducted four flights (flown by four different test pilots) to a maximum altitude of 64,000 ft, dumping cockpit pressurization at the maximum altitude and flying a controlled descent recovery profile. The PPB was set to 70 mm Hg. All flights went well, with the test pilots reporting that they hardly noticed the PPB.

At this time we declared that we had developed the requested system for the Canadian Forces. However, by this time the plans for the New Fighter Aircraft had been solidified into a commitment to the F-18. This aircraft did not have any significant altitude capability, so the CF thanked us for our efforts and lost interest in our work.

USAF Tactical Life Support System (TLSS)

The expertise developed at DCIEM was recognized, however, when the SOR for TLSS specifically directed that the TLSS altitude protection would be as developed at DCIEM. This lead to a long association with the USAF through the contractors Boeing and Gentex. DCIEM designed the TLSS G-suit and actually constructed the first integrated TLSS flight suit. DCIEM also carried out all the altitude trials and tested the complete system prior to its going to USAF SAM for the final acceptance trials.

Since TLSS

Since the completion of the TLSS project, DCIEM has continued to expand the limits of our knowledge of the physiology of positive pressure breathing with the support and collaboration of the NAWC Warminster.

We have demonstrated that G-suit coverage is very important for protection against the adverse physiological effects of PPB (Goodman, et al., 1992). The new full-coverage G-suits give far superior coverage compared to the standard G-suit. However, our understanding of the complete physiology of PPB is still incomplete especially with respect to arterial oxygen saturation (Buick & Porlier, 1991). We have also demonstrated that it is possible for subjects to sustain PPB levels up to 88 mm Hg for at least 20 minutes with complete coverage G-suits. With the standard G-suit, 5 minutes of PPB at 70 mm Hg was an eternity.

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Discussion

DR. ACKLES: Where are we going from here? We have had an idea for a long time that we can provide smart protection for aircraft of the year 2020, where the protection will be tendered according to your physiological responses. We are developing some algorithms and doing some preliminary studies to see whether we can control physiological variables within established ranges, e.g., by varying G-suit ratios or body coverage under various conditions. We are going to continue to develop these control algorithms as well as physiological modeling of the systems. We've got a good start on the models and we're hoping to continue the effort with, hopefully, some collaboration.

We also have at the DCIEM a very well-constructed database of our research for the past ten years. The raw physiological signals are all stored on optical disks, so we can go back and re-analyze things any way we desire. I feel it is very important for all of our research organizations to commonly share our in-house databases.

DR. MOON: The inflight testing was very nice work, and you now have proof of the system since you actually had pilots flying and they seemed to have no problem. On the other hand, it's clear from your earlier measurements that you're operating right near the edge.

DR. ACKLES: Yes, we are.

DR. MOON: Even on the best of circumstances you had a 15% reduction in stroke volume. Your saturations of 65 to 70%, I think most people would say were barely acceptable. Professor Ernsting's comments that even at an actual altitude of 60,000 feet, the cabin altitude may be higher than that following decompression because of the suction effect. You could make your results a very strong argument for a full-pressure suit. It seems that even using the best of equipment and the highest tolerable pressures, that some individuals just may not be able to make it under those circumstances with a partial-pressure garment.

DR. ACKLES: It is a possibility, but we felt that we've explored the limits of the partial-pressure garmentry without the need for fully pressurized helmets. We've got a lot of data that people can access for specific details, if they want.

DR. GOODMAN: I think when Dr. Ackles gives Dr. Fraser's paper, you'll see that the data gets better when we start using the full-coverage lower body suits.

DR. ACKLES: Yes, there's a big difference using full-coverage suits.

DR. SEARS: As a general question, I know you indicated that there is considerable tearing of the eye at altitudes above 60,000 feet. I have never been exposed to those altitudes without a fully pressurized helmet, but it seems to me that if you go above 63-65,000 feet, the corneal layer of the eye is going to dry out pretty rapidly if the guy doesn't tear. You've heard me wonder before whether the cornea would dry out over a 2 or 3 minute period of time, especially if the individual wasn't tearing.

DR. ACKLES: With positive pressure breathing?

DR. SEARS: Yes, but with the surface of the eye exposed to the ambient pressures.

DR. ACKLES: We have to assume you're blinking still.

DR. SEARS: Yes, you're probably still blinking.

DR. PILMANIS: We did look at the guinea pigs eyes, and did not see any effect.

DR. SEARS: As I remember, Julian Cook did see some problems with the dogs' eyes. It's something that should be kept in mind during high-altitude studies. I understand most studies have found little, if any effect.

DR. PILMANIS: Are there any more comments or questions?

MS. MCGARVEY: I have one question. In light of everybody's work, is there a significant difference between males and females in the response to high-altitude exposure?

DR. PILMANIS: From the DCS standpoint, we're in the middle of really the first prospective series of studies on comparing males and females and right now we don't have enough data to make any statement. The data that's been available for years from chamber training indicated that females are four times as susceptible as males to DCS, but there are many questions about these data and retrospective studies are no substitute for a well-designed study.

DR. ACKLES: For the physiological studies at altitude, we've used females and males interchangeably. We have not seen any gender differences.

DR. STOLP: We've also haven't noted any gender differences during studies to determine the effect of breathing sustained positive pressures of 60 mm Hg at altitude.

COL. SHERMAN: It is very important to consider whether the life-support shop, maintenance, and other ground personnel will be able to support all the many activities associated with these newer life-support systems. Will planning and training involve multiple sorties per day? In chemical protective equipment?

MAJ. NEUBECK: The operational squadrons do practice for repeated sorties.

COL. SHERMAN: Yes, I know that's what we did at Alconbury.

MAJ. NEUBECK: Ground crews are exposed to heavy tasking for a long time.

COL. SHERMAN: We finally kind of came to the realization that, in some of the situations, there were some pretty serious limitations on other groups that possibly might have broken down under a rapid sortie regeneration plan. So you need to ensure that the exercise fully covers all contingencies during the development of your overall strategy, e.g., if that happens are we going to continue to fly?

COL. HILL: At the current time, there is no chemically safe system in the USAF that will provide adequate breathing pressure for PBG to +9 Gz or PBA for altitudes to 60,000 feet. We don't use COMBAT EDGE with our chemical protective systems. The F-22 program is addressing the problem. I am hoping that the program is successful and can be retrofit into other fight aircraft. But right now, you can't combine both PBG and chemical protection.

MAJ. NEUBECK: We're limited to staying below 43,000 feet with the current chemical and biological protective system.

COL. HILL: But in your F-16s, where you routinely use COMBAT EDGE, you don't have chemical protection. They don't work together and there is no current operational requirement. So in a chemical war you either have chemical protection or high G, but not both, so our enemies could be very selective.

LT. COL. DEMITRY: Lt. Col. Clement indicated that a low key program was being conducted that would address the problem of both G and chemical protection, but nothing was noted about protection to higher altitudes. I would ask the acquisition people from their perspective how much does it cost for us to field a system and then potentially have to fix it; what is the cost of nonintegration, just a rough order of magnitude? Is that a problem or is the delta cost not a significant issue? I know when you are deriving requirements you must show return on investment and

increased operational capability and those are key. What is the cost of this approach in terms of very, very scarce acquisition dollars?

COL. HILL: Except for the F-22, there is no high-altitude program. It's receiving some laboratory attention but there is no 6-4 program for altitude protection.

COL. SHAFFSTALL: The F-22 has the only integrated protection system being developed.

LT. COL. DEMITRY: So if we want to somehow go back and incorporate high-altitude protection into our G- and chemical-protection systems, as opposed to trying to do an integrated approach, is there a price to pay for the lack of integration?

MS. MCGARVEY: I think the answer is yes and there is a price.

LT. COL. DEMITRY: Do we know what it is?

MS. MCGARVEY: No. It would require a change to the equipment that we fielded with COMBAT EDGE. I'm sure you could price out such a program and reason that it was not done correctly. The only other alternative is to see how successful the F-22 is with the systems that they're developing and then try to implement them in the current F-15s and F-16s.

LT. COL. DEMITRY: Yes. My point was just to make sure that the next time we have an opportunity to bring technologies together at some point, we should do everything in our power to complete the task. I'm not familiar with the F-22 program or the logic trail. I'm sure it was done with logic at every reasonable decision point and I suspect I would have followed the exact same trail, given the information at each point. The costs could be very high in the future, however, if we don't bring all of the needs together early on.

COL. HILL: We have to remember that there was a tactical life-support system program in the 1980s. Fortunately we're consuming that technology now so it was not a waste of time. That makes us feel very good. There was a program called the Advanced Integrated Life Support System projected for a FY 97 start that would have tied all of the protective technologies together. We're disassembling that program now because of lack of stated user need. We're taking money away from that and putting it on our Advanced Hybrid Oxygen System. Unless someone expresses a need for such a system, we will not build it. Keep in mind the using community does not pay our bills. Our best hope currently, is that there will be a retrofit. I understand that General Loh expressed interest in that approach. There is an answer to the need, but the need must be expressed.

LT. COL. DEMITRY: Yes, I just wanted that read into the proceedings. That's fine.

COL. HILL: I think that's probably one of the most critical questions you could have asked.

MS. MCGARVEY: The statement is very appropriate. The F-22 hasn't really done anything especially new; we've just used lessons learned from previous Air Force development programs.

DR. PILMANIS: In the past, fighter aircraft just didn't stay up long enough for DCS to become an issue, or they didn't have cabin altitudes high enough to be concerned about DCS. Considering future type aircraft, how much is the reduction in advanced bases and increased use of aerial refueling going to increase the exposure time of fighter pilots to higher altitudes?

MAJ. NEUBECK: Exposure time above 50,000 feet is going to be based on what the tactical situation is. I noted earlier that if you're having to cross a highly intense threat environment, then probably high altitude and high speed is the answer. If you're trying to use stealth and not be detected by the contrails, you're probably going to go high and stay high for a while. If you have to do a lot of aggressive maneuvering then you're going to come down because you can't stay high anymore. But, I really can't give you a more than a qualitative answer. The F-22 is going to expose guys to longer durations of flight above 50,000 feet. It's just a fact. They're going to go up there.

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DR. PILMANIS: Compared to previous scenarios?

MAJ. NEUBECK: Yes. If you tell a pilot he can go to 60,000 feet, he's going to go 60,000 feet.

DR. SEARS: Or to 70,000 feet.

MAJ. NEUBECK: That's right, he's going to go as far as he can go.

COL. HILL: You indicated about 400 miles this morning, which is 30 minutes or more. That's a very long time.

DR. SEARS: The vulnerability and threat people at Northrop wanted a pressure assembly that would protect to 70,000 feet for half an hour. However, they felt it was probably acceptable to come down to 50,000 feet for this 30 minute period after loss of pressure at higher altitudes.

MAJ. NEUBECK: So we have to do more studies. We have to do a fairly intensive study I think to give you a quantitative answer on time and distance. I'm pulling the 400 miles out of my pocket.

COL. HILL: I understand. It's a reasonable estimate. It's not two minutes and it's not three hours.

MS. MCGARVEY: I think your overall expected mission length is no different than what you would envision for a F-15 or F-16 currently.

MAJ. NEUBECK: That's true. Mission lengths in Desert Storm were commonly up to 6 hours. You go to a tanker and you've got bags. If you're planning on going long distances, the tankers can help get you there and then get you back home. That won't be any different for the F-22.

DR. PILMANIS: So a fighter pilot would commonly be in the cockpit for up to six hours?

MAJ. NEUBECK: Yes for six hours, but not at 60,000 feet.

MAJ. DIESEL: With potentially repeated exposures to flight altitudes of 60,000 feet?

MAJ. NEUBECK: Yes. I think Lt. Col. Demitry made a really good point this morning about flicking switches and prebreathing 100% oxygen. The F-18 aircrew fly with 100% oxygen all the time. USAF aircrew don't do that and you shouldn't put the pilot in the loop to force him to flick a switch to start breathing 100% oxygen. The environment is so dynamic that he could be transitioning from 40,000 feet to 60,000 feet in several minutes. He is definitely too busy to think about switching to 100% oxygen. So you need to take the pilot out of the loop, because he's going to make a mistake and then he might be in trouble.

DR. PILMANIS: Which brings us back to one of the main controversies, i.e., the tradeoff between use of 100% oxygen to prevent decompression sickness and use of an airmix to prevent acceleration atelectasis.

MS. MCGARVEY: The F-22 system goes into a high cycle mode if the cabin goes above 11,000 feet, which occurs at a flight altitude of 30,000 feet. So at 30,000 feet the pilot will be breathing at the maximum OBOGS oxygen concentration. So we basically have automatic switching to 100% product gas at 30,000 feet. We asked the laboratory early on if they were concerned about G-induced atelectasis above 30,000 feet and found they were more interested in ensuring that the pilot receive a higher concentration from the OBOGS to prevent hypoxia at, and above, that altitude. So we were able to simplify the OBOGS design to either a low or a high cycle.

DR. SEARS: How about purge valves? How do you get rid of the lower concentration of OBOGS product gas in the long line to the regulator following loss of pressure? Has that been considered?

MS. MCGARVEY: Yes, we have two relief ports on the system, one at the emergency oxygen regulator and another one at the BRAG valve itself.

DR. SEARS: So the lower concentrations are purged following a loss of pressure?

MS. MCGARVEY: Yes. It will not purge the entire line though. I'll answer that in more detail during my presentation. In essence, some of the line length acts as a plenum in the system.

PROF. ERNSTING: The atelectasis controversy relates very much to different philosophies from our operational staffs. One of the things we have to ask is that if you lose cabin pressure at high altitude, what do you do with the airplane? The Royal Air Force philosophy is that you come down immediately. You come down as fast as you can to below 40,000 feet and then ideally, if the distance back to base allows you, you come down to 25,000 feet aircraft altitude. The Eurofighter breathing systems will allow you to stay to 35,000 feet. I'm not absolutely sure where that requirement came from, but Dr. MacMillan and I don't believe it came from the Royal Air Force staff; there are three other nations involved in the development program. We believe that if you've been breathing airmix before a rapid decompression to 60,000 feet, and descend after a 3 or 4 minute exposure, your risk of decompression sickness is very small. We've all done it many times ourselves and have occasionally ended up with some symptoms. I must emphasize again that breathing lower oxygen concentrations to reduce atelectasis was not a medical decision; it was an operational staff decision. I think there's room for compromise in this area and we do need to come to an agreement or design new experiments to allow us to come to an agreement. There shouldn't be any difference of aeromedical or physiological opinion across the pond. The experimental results should tell us the same thing. Whether the air staff dismiss it is another problem. If the US Navy were here they would likely say they never had a problem. But, you go down to squadron level and talk to aircrew and you get a different story. I've argued about that with Navy representatives for the last 15-20 years.

COL. HILL: We must remember that we're managing risk and I hope you folks who actually fly airplanes realize that we know there are times during flight that if something goes wrong you're in a great deal of trouble. We have ejection seats today that, if you choose to initiate them during certain parts of the flight profile, you will not survive. So there may be a situation where if you choose to fly at 60,000 feet you're at risk and there's not a whole lot you can do about it with the systems you were provided. Regardless of your concept of operations, there will be a real-time decision made upon loss of pressure at high altitude that will probably be a compromise between a physiological hazard and an enemy threat. But we have to be practical enough to know that we can't protect against all situations all the time. Just so the people who wear wings know, we know you're not going to accept a full-pressure suit in air-to-air combat. Unless there is a revolution in the way we build pressure suits, you will not accept it. We understand that. However, we're looking at the outer boundaries. We know what's important when you fly and that's being able to move and see, and you can't move and see in a current full-pressure suit. I'd be willing to take comments on it.

DR. BOMAR: I can endorse that. What do you believe is our obligation to develop effective methods of communicating the risks to aircrew in a way that they can make those decisions intelligently?

COL. HILL: There's no good answer to that, but indeed if we've got a charter somewhere, it's simply presenting technology options. I think that's probably the reason we're here today. You must understand there may be some operational decrement if we fix the problem, or you just accept that risk. If we choose to keep the systems unintegrated, and that's what we've chosen to do today, then there's a cost that goes along with it.

It's very easy for people building the aircraft today to ask us to investigate the entire envelope. If at least one or more of the research community is not briefed into the program we can't be expected to know the absolute limits of the system. We can't spend the money and we can't put people at risk unless we know that there is an operational need. Even then, some of the more hazardous conditions will not be approved by our human use committee. We don't have a choice; we need to know where you're going to fly. In fact, I've got to write a talking paper next week to the director of the Armstrong Laboratory on that very subject, on what do we need to look at because there's going to be a cost and we may not be able to afford it. I don't know if that answers your question.

DR. BOMAR: I just don't think we ever communicated very effectively with the aircrew in a way that allows them to help us make the decision.

COL. HILL: I couldn't agree more. You're absolutely right.

DR. BOMAR: It's partially our fault. We sit around here and talk about hypocapnia and PCO_2s and PAO_2s and there's an aircrew member in the room. You may be a physiologist for all I know?

MAJ. NEUBECK: Some of it is going over my head.

DR. BOMAR: I think we need to explain the real consequences in terms of performance to aircrews so they can help make the decisions.

DR. ACKLES: I think this question of risk analysis brings back memories of discussions we had before doing our 80,000 feet studies. We realized we would be really on the edge at 80,000 feet and seriously questioned the operational need. We asked the operational staff at least three separate times whether they were really sure that 80,000 feet was a requirement. The air staff indicated that it was necessary to get to 80,000 feet to release missiles in a parabolic maneuver. We spent a lot of time considering total flight times at various altitudes, chance of losing pressure, other contingencies and physiological risks. We felt that even if they were at 80,000, we'd give them a chance they didn't have before, by developing a pressure ensemble similar to that the UK had used successfully to 60,000 feet. One of the primary goals of the developmental system was to provide short-term protection to 80,000 feet without encumbering them with the same protection that would be required if they were to remain at 80,000 feet, like a full-pressure suit. It was a tradeoff that we felt was necessary. We were required to use 80 mm Hg breathing pressures at this altitude in order to give them a chance they wouldn't have with any other system.

SECOND DAY--OPENING COMMENTS

DR. PILMANIS: We have spent a lot of time discussing a 5 PSI cabin pressurization schedule. The schedule is given in Mil-E-18927. This is the same schedule that is in the F-22. And the Eurofighter?

PROF. ERNSTING: Yes.

DR. PILMANIS: We also were discussing whether aircraft were currently flying at altitudes above 50,000 feet. I have an article, entitled, " Don't Stretch Your Limits." An F-16 pilot went to 86,000 feet without knowing that there was a onboard recorder. He just took it for a spin. The engine flamed out, he lost pressurization and fell out of the sky. Luckily he recovered at 20,000 feet, landed and walked away. The article was written by the chief test pilot at Lockheed. Here is some documentation that they fly above 50,000 feet.

The Physiology of Pressure Breathing

B.W. Stolp, M.D., Ph.D.
R. E. Moon, M.D.

INTRODUCTION

Positive pressure breathing (PPB) is used to maintain the alveolar partial pressure of oxygen during altitude exposures in excess of 12,000 meters (36,000 feet). At altitudes up to 33,000 feet, the alveolar gas tensions may be kept within the normal range by increasing the concentration of oxygen in the inspired gas mixture. Above this altitude, however, alveolar oxygen tensions fall below normal levels despite breathing 100% oxygen (Sharp & Ernsting, 1988). Furthermore, during high-acceleration (high-G) maneuvers, even at lower altitudes, there is a tendency for microatelectasis formation and development of intrapulmonary right-to-left shunt, which may be accentuated during 100% oxygen breathing (Wagner, et al. 1977). Raising the pressure inside the breathing circuit throughout the breathing cycle (PPB) is well established as a method of maintaining alveolar oxygen tension at high altitudes (Gagge, et al. 1945) and under high G-forces. Burns and Balldin (1988) studied the rates of arterial desaturation during acceleration with positive pressure breathing at 50 mmHg and 70 mmHg. They found that the rate of oxygen desaturation was less with PPB than without PPB suggesting a possible improvement in $\dot{V}_A/\dot{Q}$ relationships, at least in low $\dot{V}_A/\dot{Q}$ regions or shunt. This is equivalent to continuous positive airway pressure (CPAP) used clinically to augment arterial oxygenation in patients.

PPB may have detrimental effects, however. Increased airway pressures can cause pharyngeal discomfort with throat and neck distention, as well as creating difficulties in the maintenance of a tight mask seal. The increased intrapulmonary pressure can result in lung hyperexpansion. This is minimized by the use of a counterpressure vest (Burns and Balldin, 1988). The increase in intrathoracic pressure also results in diminished cardiac output due to decreased venous return from peripheral capacitance vessels and may eventually induce syncopal symptoms (Goodman et al, 1992; Ackles et al, 1978; Balldin & Wranne, 1980). In patients treated with CPAP, reduced cardiac output and renal blood flow have also been observed. PPB at 30 mmHg without chest counterpressure and at 60 mmHg with counterpressure causes increased tidal volume, minute ventilation, oxygen consumption and carbon dioxide elimination (Ernsting, 1977). While nitrogen clearance curves indicate that the distribution of inspired gas in the lung is minimally changed with PPB, Fenn et al (1947) estimated that nearly 50% of the blood in the lung is displaced during PPB at slightly over 20 mmHg. It is, therefore, likely that PPB causes an increase in high ventilation perfusion ($\dot{V}_A/\dot{Q}$) areas in the lung, and hence impairment of carbon dioxide exchange, providing an explanation for the hyperpnea (and hence increased work of breathing) that usually results.

The purpose of this study was to investigate the cause of the well-described hyperventilation that is associated with moderate to high levels of PPB. It was hypothesized that PPB with both chest counterpressure and leg compression from an anti-G suit reduces perfusion to low $\dot{V}_A/\dot{Q}$ units in the lung while increasing the ventilation of high $\dot{V}_A/\dot{Q}$ units. It was also hypothesized that microatelectasis induced by 100% oxygen breathing (Wagner et al, 1974) would be reduced by PPB. The multiple inert gas elimination (MIG) technique was used to quantify the changes in $\dot{V}_A/\dot{Q}$ relationships during PPB using the COMBAT EDGE that uses positive pressure breathing, a chest counterpressure vest, and an anti-G suit.

METHODS

Ten healthy male non-smoking volunteers aged 20 to 33 years with normal lung function and good exercise tolerance were selected for the study. The risks of positive pressure breathing, pulmonary and arterial catheterization, and altitude exposure were explained and informed consent was obtained in accordance with the guidelines of the Duke University Institutional Review Board for Human Experimentation (Protocol #1204-93-9).

Prior to the experiment each subject underwent a series of 1-3 training sessions in order to become familiar with the equipment and to become accustomed to maintaining steady-state breathing patterns at both 30 and 60 mmHg mask pressure for a minimum of five minutes on the COMBAT EDGE.

Experimental Set Up

All studies were performed in "F" chamber at the F.G. Hall Environmental Laboratory at Duke University Medical Center. The subject was seated in a semi-reclined position to simulate the position of the pilot in an F-16 during flight. The CRU-93 pressure demand regulator supplied gas to the breathing mask, the chest counterpressure vest and anti-G suit. Breathing gas pressures of 0 mmHg, 30 mmHg, or 60 mmHg were provided according to the experimental protocol (see below). Pressure was either provided to the mask alone or in a 1:1 pressure ratio to the mask, the upper body counterpressure garment, the helmet bladder, and the G-suit as outlined.

Expired gas was conducted through a heated expiratory tube and mixing box into a Douglas bag. Expired bag volume was measured with a calibrated gasometer. Appropriate temperatures and barometric pressures were recorded for later use in conversion of gas volumes to either STPD (metabolic studies) or BTPS (ventilatory studies).

Analog signals for mask pressure, central venous (CVP), pulmonary artery (Ppa), and arterial pressures (Part) were conducted via through-hull penetrators to a standard patient monitor. These signals were also digitized for data storage and analysis.

A dilute solution of inert gases for determination of ventilation perfusion relationships according to the method described by Wagner et al (1974) was infused by a central venous catheter inserted via the contralateral basilic vein.

Cardiopulmonary Measurements

Throughout the entire day the subject's condition was monitored continuously by a physician outside the altitude chamber for fatigue, mental status changes, electrocardiographic changes, and blood pressures. Digitized data were collected during the ten minutes before and after each study. These were later analyzed for heart rate, systolic and diastolic arterial blood pressure, mean arterial, pulmonary artery, central venous and mask pressures, maximum and minimum mask pressures, and respiratory rate. Pulmonary capillary wedge pressure (PCWP) and central venous pressure (CVP) were also recorded at end expiration for later analysis. These values were taken over at least three consecutive respiratory cycles in order to assure reproducible measurements. Wedge pressures and CVP pressures measured at end expiration were discarded if the subject experienced a mask leak during measurement or was unable to maintain a constant mouthpiece pressure. Heart rates were averaged over a 10-20 second period during each minute of the experiment. Corresponding blood pressure measurements were obtained over the same time period.

Cardiac output. Fick cardiac outputs were determined during each experiment using 3 of the 6 inert gases and oxygen. Thermodilution cardiac outputs were also obtained in the last 8 subjects. Thermodilution curves were repeated until either the subject became fatigued, was unable to maintain a good mask seal, or until two consecutive cardiac outputs were reproducible.

Minute ventilation. Minute ventilation values are reported as BTPS using ambient chamber barometric pressure. During runs at increased mask pressure, when intrathoracic pressure exceeds ambient pressure, the usual calculations of BTPS ventilations therefore slightly overestimate the volume of gas moved through the breathing

circuit. This does not affect blood gas measurements, or calculations of oxygen and carbon dioxide exchange. The effect on dead space is discussed below.

Metabolic Calculations: $\dot{V}$ O_2 (oxygen consumption), $\dot{V}$ CO_2 (carbon dioxide elimination rate) and R (respiratory quotient) during air breathing experiments were calculated according to standard steady state equations. Dead space was calculated using the Enghoff modification of the Bohr equation.

P_ECO_2 is conventionally derived from the ambient pressure and the fractional concentration of carbon dioxide in each mixed expired gas sample. However, when mask pressure is elevated the entire breathing circuit and tracheobronchial tree are at increased ambient pressure (Pbar + 30 or Pbar + 60 mmHg). Compared to values calculated using ambient (chamber) pressure, dead space calculated using mask pressure is therefore somewhat lower. When mask pressure is 60 mmHg the downward adjustment of dead space at ground level is around 16% and at 24,900 feet altitude, it is 30%. Thus, adjusted values for dead space were also calculated (see Summary of Blood Gases and Ventilation in Appendix). All reported blood gas tensions reflect the tensions within a liquid and therefore do not require an adjustment for mask pressure.

Statistics

In order to test the effect of experimental condition, 3-factor ANOVA was used. Statistical significance was defined as $P < 0.05$. When statistically significant effects were observed, post-hoc paired comparisons were made using the Tukey-Kramer test.

Experimental Protocol

The matrix of experimental conditions is shown in the following table.

Altitude (feet)	FiO_2	Mask Pressure (mmHg)	G-suit Pressure (mmHg)	Thoracic Counterpressure (mmHg)
460	0.21	0	0	0
460	0.21	30	30	30
460	0.21	30	0	0
460	0.21	60	60	60
460	1.0	0	0	0
460	1.0	60	60	60
24,900	1.0	0	0	0
24,900	1.0	60	60	60

The six ground-level runs were conducted at ambient barometric pressure (P_B) with the chamber door open (mean P_B = 756 mmHg, range 741-764 mmHg). For altitude measurements, the chamber was decompressed at a rate of 2500 ft/min to a simulated altitude of 24,900 feet (P_B = 282 mmHg). This altitude was chosen as a compromise between minimizing oxygen prebreathing requirements needed to decrease decompression sickness risk for inside personnel and an attempt to maximize simulation of high-altitude gas density and hypoxia. Prior to going to altitude, all chamber occupants breathed 100% oxygen for at least 30 minutes in order to minimize the possibility of decompression illness. They remained on oxygen throughout the altitude exposure and during the return to the ground level. The descent rate was similar to the ascent rate with stops made when necessary if significant middle-ear discomfort was noted by the inside personnel. After each change in altitude the chamber temperature was allowed to stabilize before beginning the pressure and volume calibrations prior to the next run. Each experimental period lasted 3.5 - 12 minutes. The minute-by-minute protocol is summarized as follows. The target pressure was delivered following pressure and volume calibration and verification of adequate electrical signals on the data acquisition system. The experimental clock was started following attainment of steady-state minute ventilation, breathing pattern, heart rate and blood pressure. Minute ventilation was monitored on-line from an Ohmeda 5410 volume monitor in the chamber while the remainder of physiological parameters were monitored by the medical officer outside the chamber. The Douglas bag was opened and duplicate arterial and mixed venous inert gas blood samples were obtained after two minutes of steady state breathing. The

corresponding inert gas mixed expired sample was collected from the mixing box after real-time calculation of the transit time between the mouth and expired gas sampling port. Inert gas (MIG) blood samples were generally obtained by constant aspiration over the course of one minute. However, during 60 mm mask pressure runs, which were often limited by subject tolerance, 30 second samples were usually obtained. Separate arterial and venous blood samples were drawn for analysis of hemoglobin (Hb), hematocrit (Hct), partial pressure of arterial and mixed venous oxygen and carbon dioxide (PaO_2, $PaCO_2$, $P\bar{v}O_2$, $P\bar{v}CO_2$), arterial and mixed venous pH (pHa, pH$\bar{v}$), and arterial and mixed venous oxygen saturation (SaO_2, $S\bar{v}O_2$). The blood volume collected for each run was approximately 30 ml; the total volume was less than 300 ml/day.

After all blood samples had been drawn and the lines flushed, 2-4 thermodilution cardiac outputs were obtained. Pulmonary artery wedge pressure measurements were obtained by inflating the balloon on the pulmonary artery catheter until the wedge wave form was verified by the outside medical officer. The balloon remained inflated over several respiratory cycles to provide reproducible end-expiratory measurements. Each experimental period was stopped after all samples and measurements had been obtained, when the subject indicated that he could not continue any longer, or when pre-syncopal symptoms were noted.

The order of experimental conditions was randomized within certain constraints. The second subject (KK) developed severe abdominal distress at altitude after performing a 60 mmHg run at ground level. Subsequent altitude studies always preceded ground-level pressure runs of 60 mmHg in order to minimize the intake of gastrointestinal gas at ground level which, because of expansion, could then induce discomfort during ascent.

Data Analysis

Inert Gas Calculations: Under each experimental condition, for each inert gas, retention ($Pa/P\bar{v}$) and excretion ($PE/P\bar{v}$) were calculated. With a FORTRAN program on a Gateway personal computer, using the method of ridge regression (Wagner et al, 1974), a 50-compartment distribution of ventilation and perfusion was obtained. Shunt (Q_s/Q_t: $\dot{V}_A/Q$ = 0) and dead space (V_D/V_T: $\dot{V}_A/Q$ = _) represent the extremes of this distribution. Log standard deviations of the ventilation and perfusion distributions (logSD$\dot{V}$, logSD$\dot{Q}$) provide indices of ventilation-perfusion mismatching (Evans & Wagner, 1977). Additional parameters obtained directly from the inert gas measurements, without requiring the 50-compartment distribution, were calculated as described by Gale et al, 1985.

RESULTS

The experimental results are presented in the Appendix and summarized as follows. Minute by minute data was acquired as described under the section "Cardiopulmonary Measurements". Graphs of mean data comparing the various conditions represent steady-state values obtained during the last minutes of each run.

Mean mask pressure was maintained within 4 mmHg of target pressure during all experimental runs. There was a significant effect of PPB pressure on $\dot{V}_E$ ($P < .0001$) (Figure 1). Individual paired comparisons indicated that each of the three pressure conditions was significantly different from zero mask pressure ($P < .05$). There was no significant effect of breathing gas or altitude on minute ventilation.
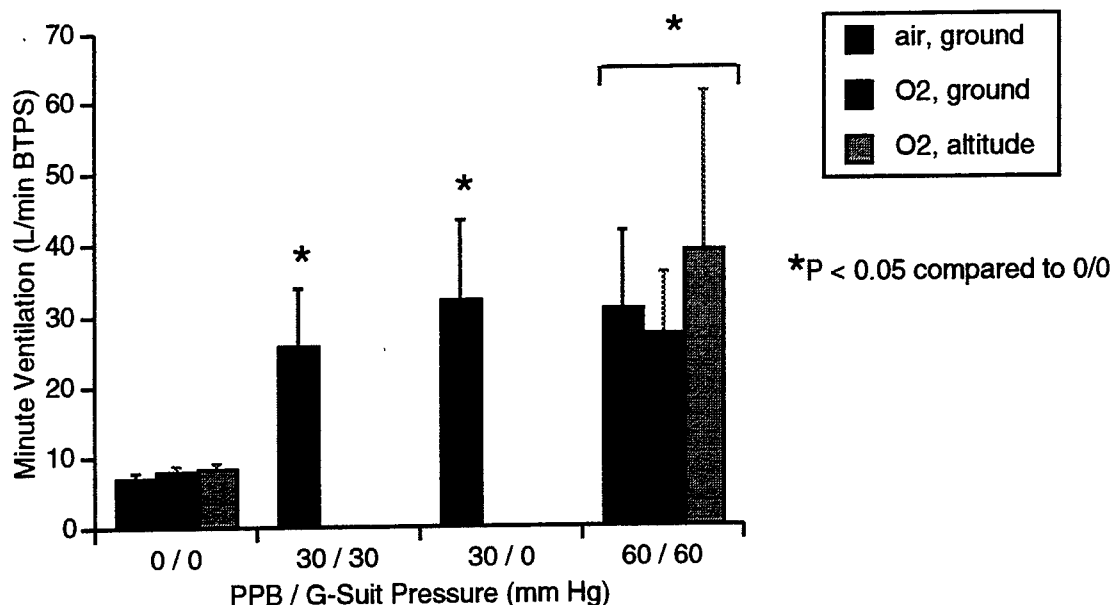
Figure 1: Minute ventilation vs. condition.

There was no significant effect of pressure condition on oxygen uptake. Carbon dioxide elimination rate increased significantly under all conditions of increased mask pressure (range 189 to 485 ml/min) ($P < .0001$). Each of the conditions with increased mask pressure was significantly different from zero mask pressure ($P < .05$). The increased carbon dioxide elimination mostly represents the effects of acute hyperventilation, rather than increased metabolic carbon dioxide production. There was no significant effect of breathing gas or altitude on carbon dioxide elimination.

Increased mask pressure resulted in a significant reduction in arterial PCO_2 ($P < .0001$) (Figure 2). Each of the increased mask pressure conditions was significantly different from zero mask pressure. There was no significant effect of breathing gas or altitude on arterial PCO_2.
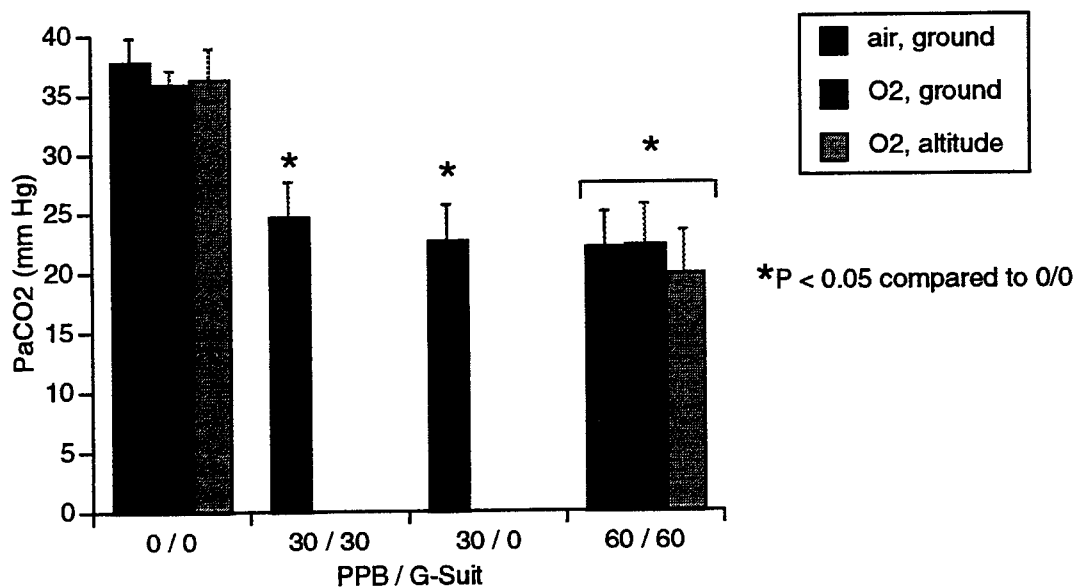
Figure 2: $PaCO_2$ vs condition.

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Increased mask pressure resulted in significant increase in arterial pH (P < .0001) (range 7.39 to 7.62). Each of the increased mask pressure conditions was significantly different from zero mask pressure (P < .05). There was no significant effect of altitude or breathing gas on arterial pH. The change in arterial pH was entirely due to the effects of reduced arterial PCO_2.

Both breathing gas and altitude significantly affected arterial oxygen tension (P < .0001). As expected from the alveolar gas equation, reduction in arterial PCO_2 during PPB was associated with a rise in PO_2 (P < .0001).

Pulmonary Gas Exchange Efficiency Measurements

Bohr V_D/V_T ratio increased at altitude compared to ground level (P < .025). However, when V_D/V_T was adjusted for mask pressure there was no significant effect of altitude (see Appendix). There was no significant effect of either breathing gas or mask pressure on V_D/V_T. However, increasing mask pressure resulted in a significant increase in tidal volume (P < .0001) (range 0.622 to 2.477 L/BTPS). There is no effect of mask pressure on ventilatory frequency (see Appendix), although two individuals became tachypneic toward the end of the 60 mm PPB runs with ventilatory rates in the 40s, consistent with respiratory muscle fatigue.

Dead space calculated by the Enghoff modification of the Bohr equation significantly increased with increasing mask pressure (P < .0001) (0.204 to 0.930 L BTPS). All three conditions of increased mask pressure were significantly different from zero mask pressure (P < .05). The increase in CO_2 dead space was due to increased tidal volume. During PPB, the unchanged V_D/V_T ratio and reduced $PaCO_2$ provide evidence that the marked hyperpnea that the subjects exhibited was not merely compensation for increased wasted (dead space) ventilation.

Inert Gas Analysis and $\dot{V}_A/\dot{Q}$

During positive pressure breathing there is a progressive shift to the right of the $\dot{V}_A/\dot{Q}$ distribution, reflecting both perfusion and ventilation shifting to lung units with higher $\dot{V}_A/\dot{Q}$ ratios. Adjustment of mixed expired inert gas partial pressures in a manner similar to the adjustment for P_ECO_2 would tend to minimize these changes. Shunt is zero during air breathing. There is no effect of pressure breathing on dead space as a fraction of tidal volume. During oxygen breathing some runs were associated with a slight increase in shunt and perfusion of low $\dot{V}_A/\dot{Q}$ units. This was particularly evident at altitude, since this experimental run was preceded by at least 60 minutes of O_2 breathing. High mask pressure tended to eliminate these changes, though the effect of PPB on perfusion to units with $\dot{V}_A/\dot{Q}$ less than 0.1 did not reach statistical significance. There was, however, a significant decrease in perfusion of lung units with $\dot{V}_A/\dot{Q}$ ratio between 0.1 and 1.0. The changes in $\dot{V}_A/\dot{Q}$ during O_2 breathing and at altitude are similar to those seen during air breathing at ground level, although dead space was significantly increased at altitude as noted above.

Though the ventilation to lung units with $\dot{V}_A/\dot{Q}$ ratio > 1 tended to increase at increased mask pressure, there was no significant effect on dead space. This is confirmed by the fact that $DISP_E$ was not significantly affected by any of the independent variables, including mask pressure (see Appendix). This same type of adjustment for mask pressure could be performed on the inert gas measurements. This would serve only to further minimize the statistically significant, but relatively small, physiological changes.

Increased mask pressure induced a shift of perfusion and ventilation to higher $\dot{V}_A/\dot{Q}$ units. Higher mask pressure also induced an increase in the log standard deviation of the perfusion distribution. At 60 mmHg mask pressure, this parameter increased approximately two-fold while breathing air at ground level and about 50% at altitude.

$\dot{V}_A/\dot{Q}$ was also affected by altitude. At altitude there was increased perfusion of lung units with low $\dot{V}$ A/Q and shunt, and also increased ventilation of high $\dot{V}_A/\dot{Q}$ units and dead space. Because of the necessity of oxygen prebreathing it is impossible to exclude the fact that these changes may have represented the cumulative effect of 100% oxygen breathing rather than altitude *per se*.

Hemodynamic Measurements

Minute-by-minute trends of the various hemodynamic variables are plotted below with statistical comparisons summarized in Tables 1 to 3.

Figure 3 shows the progressive increase in heart rate with increasing mask pressure (P < .0001). Heart rate at each of the increased mask pressure conditions was significantly different from the zero mask pressure condition (P < .05). Two subjects demonstrated a bradycardia associated with inability to maintain pulse pressure and mean arterial pressure prior to terminating the 60/60 mm Hg run.
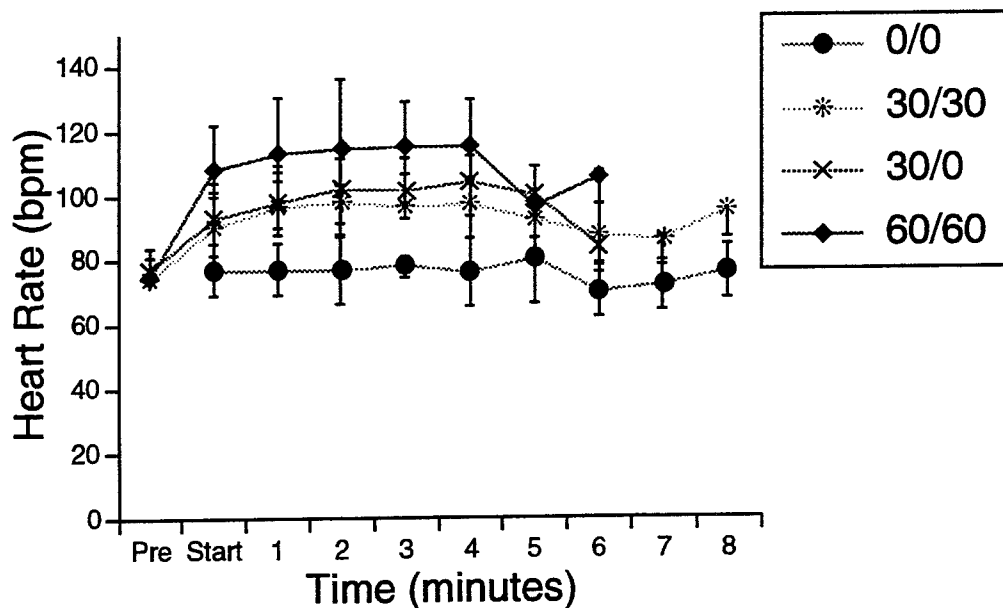
Figure 3: Heart Rate Vs. Time.

Figure 4 shows that mean arterial pressure progressively increased with increasing mask pressure (P<.0001).
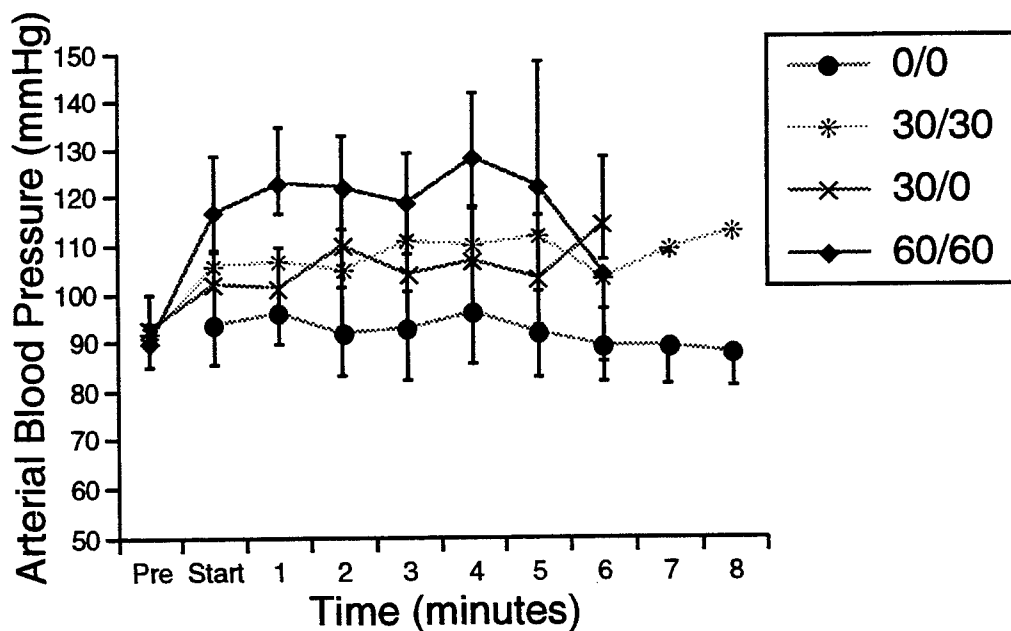
Figure 4: Arterial blood pressure vs. time.

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TABLE 1: P VALUES - CARDIOPULMONARY MEASUREMENTS

Variable	FACTOR (ANOVA)				STATISTICALLY SIGNIFICANT COMPARISONS ($P < .05$)		
	Altitude	FiO_2	Mask Pressure Condition	Pressure-Altitude Interaction	30/0 vs.	30/30 vs.	60/60 vs.
Minute Ventilation ($\dot{V}_E$)	NS	NS	.0001	NS	0/0	0/0	0/0
Ventilatory Frequency (V_f)	NS	NS	NS	NS			
Tidal Volume (V_T)	NS	NS	.0001	NS	0/0	0/0	0/0
O_2 Consumption ($\dot{V}O_2$)	-	-	NS	NS			
CO_2 Elimination ($\dot{V}CO_2$)	NS	NS	.0001	NS	0/0	0/0	0/0
Arterial PO_2 (PaO_2)	.0001	.0001	.0001	NS	0/0, 60/60	0/0, 60/60	0/0, 30/0, 30/30
Arterial PO_2 ($PaCO_2$)	NS	NS	.0001	NS	0/0	0/0	0/0
Arterial pH (pHa)	NS	NS	.0001	NS	0/0	0/0	0/0
Mixed Venous O_2 Saturation ($S\bar{v}O_2$)	.0001	.0001	.0001	NS	0/0	0/0	0/0
CO_2 V_D/V_T Ratio at $P_{Ambient}$ (V_D/V_T: P_{Amb})	.025	NS	NS	NS			
CO_2 Dead Space at $P_{Ambient}$ (V_D: P_{Amb})	.036	NS	.0001	NS	0/0	0/0	0/0
CO_2 V_D/V_T Ratio at P_{Mask} (V_D/V_T: P_{Mask})	NS	NS	NS	.058			
CO_2 Dead Space at P_{Mask} (V_D: P_{Mask})	NS	NS	.0001	NS	0/0	0/0	0/0
O_2 Fick Cardiac Output (CO-O_2)	-	-	.032	NS			0/0
MIG Fick Cardiac Output (CO-MIG)	NS	NS	.029	NS			0/0
Thermodilution Cardiac Output ($C\bar{O}$-TD)	NS	NS	NS	NS			0/0

Probability values calculated using 3-way ANOVA. Individual comparisons performed using Tukey-Kramer test.

TABLE 2: P VALUES - HEMODYNAMIC MEASUREMENTS

Variable	FACTOR (ANOVA)				STATISTICALLY SIGNIFICANT COMPARISONS (P < .05)		
	Altitude	FiO$_2$	Pressure Condition	Pressure-Altitude Interaction	30/0 vs.	30/30 vs.	60/60 vs.
Mouthpiece Pressure	NS	NS	.0001	NS	0/0, 60/60	0/0, 60/60	0/0, 30/0, 30/30
Heart Rate	.0084	NS	.0001	.063	0/0	0/0, 60/60	0/0
Arterial Pressure	.0043	NS	.0001	.0023	0/0, 60/60	0/0	0/0
PA Pressure	NS	NS	.0001	NS	0/0, 60/60	0/0, 60/60	0/0, 30/0, 30/30
CVP	NS	NS	.0001	NS	0/0, 60/60	0/0, 60/60	0/0, 30/0, 30/30
Systolic BP max	.0022	NS	.0051	.023		0/0	0/0
Diastolic BP max	.03	NS	.0001	NS	0/0, 60/60	0/0, 60/60	0/0, 30/0
Systolic BP min	.007	NS	NS	.014			
Diastolic BP min	NS	NS	.0001	.05		0/0	0/0
Pulse Pressure max	.056	NS	.04	NS			0/0
Pulse Pressure min	.007	NS	.0001	NS	0/0		0/0

Probability values calculated using 3-way ANOVA. Individual comparisons performed using Tukey-Kramer test.

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TABLE 3: P VALUES - MULTIPLE INERT GAS MEASUREMENTS

Variable	FACTOR (ANOVA)				STATISTICALLY SIGNIFICANT COMPARISONS (P < .05)		
	Altitude	FiO$_2$	Pressure Condition	Pressure-Altitude Interaction	30/0 vs.	30/30 vs.	60/60 vs.
$\dot{Q}(\dot{V}_A/\dot{Q}=0)$	.023	NS	NS	.023			
$\dot{Q}(\dot{V}_A/\dot{Q}\ 0-.01)$	.028	NS	NS	NS			
$\dot{Q}(\dot{V}_A/\dot{Q}\ .01-.1)$	.037	NS	NS	NS			
$\dot{Q}(\dot{V}_A/\dot{Q}\ .1-1)$	.029	.029	.0001	NS	0/0	0/0	0/0
$\dot{Q}(\dot{V}_A/\dot{Q}\ 1-10)$	.0002	.032	.0001	NS	0/0	0/0	0/0
$\dot{Q}(\dot{V}_A/\dot{Q}\ 10-100)$	.003	NS	.020	.004			
$\dot{V}(\dot{V}_A/\dot{Q}\ 0-.01)$	NS	NS	NS	NS			
$\dot{V}(\dot{V}_A/\dot{Q}\ .01-.1)$	NS	NS	NS	NS			
$\dot{V}(\dot{V}_A/\dot{Q}\ .1-1)$	NS	.01	.0001	.019	0/0	0/0	0/0
$\dot{V}(\dot{V}_A/\dot{Q}\ 1-10)$	.002	NS	.0001	NS	0/0	0/0	0/0
$\dot{V}(\dot{V}_A/\dot{Q}\ 10-100)$	.0007	NS	.012	.012			0/0
$\dot{V}(\dot{V}_A/\dot{Q}=\infty)$	.025	NS	NS	NS			
logSD$\dot{Q}$	NS	NS	.0001	NS	0/0		0/0
logSD$\dot{V}$	NS	NS	NS	NS			
DISP$_R$	NS	NS	.030	NS			
DISP$_E$	NS	-NS	NS	NS			
DISP$_{R-E}$	NS	NS	NS	NS			

Probability values calculated using 3-way ANOVA. Individual comparisons performed using Tukey-Kramer test.

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Despite the elevated arterial pressure, pulse pressure progressively decreased with duration of the experiment run (P = .019) (Figure 5). The progressive reduction in pulse pressure most likely represented the cumulative effect of reduced venous return over time. In some individuals, this resulted in reduced cardiac output and evidence of reduced cerebral blood flow, manifested by loss of consciousness.
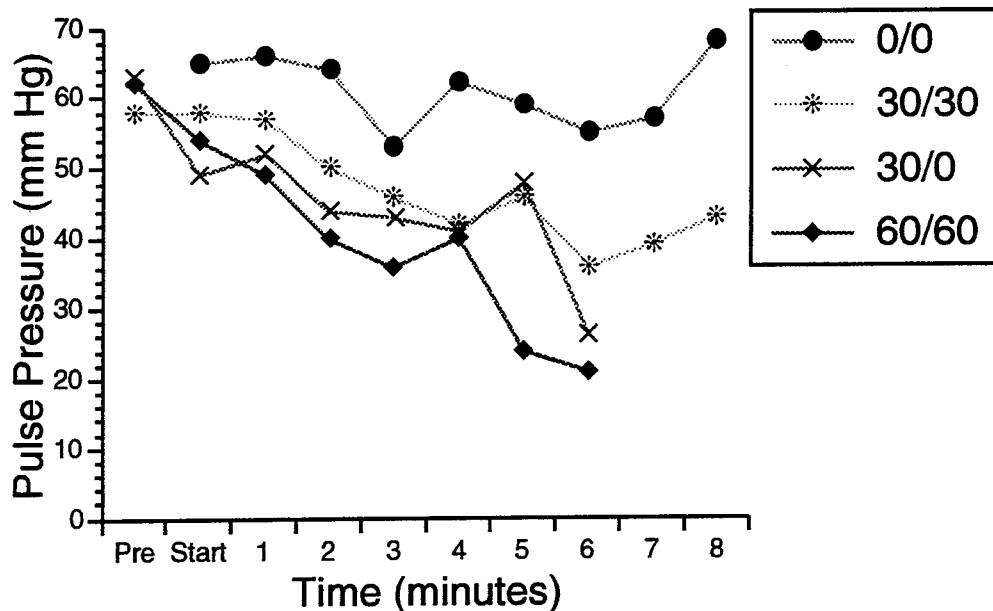
Figure 5: Pulse pressure vs. time.

The high mask pressure runs were associated with a cyclical variation in blood pressure in concert with respiratory movements. Figure 6 shows the raw data from one of the 60 mm Hg runs. The top tracing is the arterial blood pressure and the bottom tracing is the mask pressure. Both the absolute values of the blood pressure as well as the difference between systolic and diastolic pressure (pulse pressure) varies with mask pressure This probably represented pulsatile changes in venous return during the respiratory cycle.
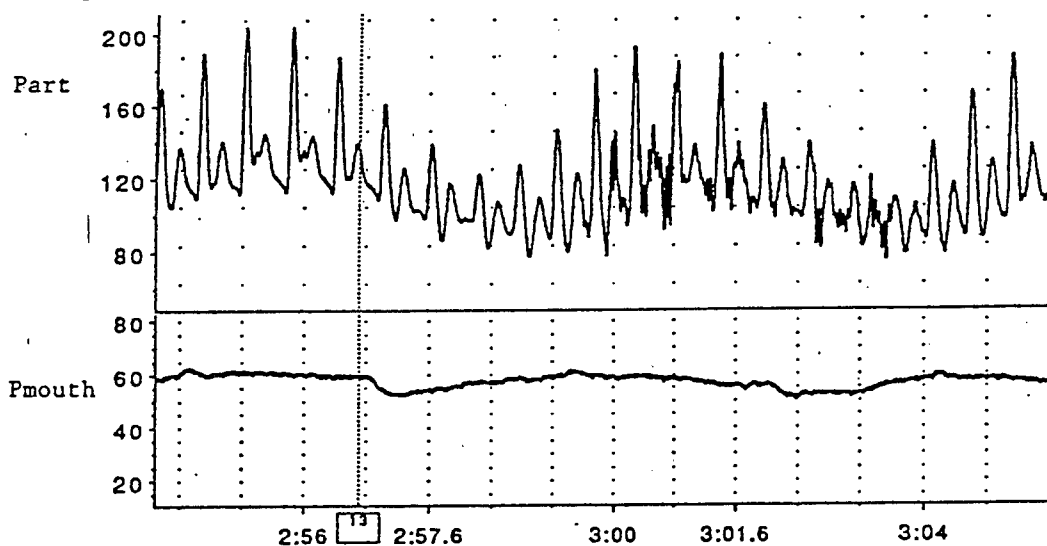
Figure 6: Example of cyclical variation in arterial pressure during 60 mmHg pressure exposure. SB breathing air at ground level.

Figures 7 and 8 depict the respiratory variation in pulse pressure as a function of time. The gradual reduction in both "maximum" (top point) and "minimal" (bottom point) pulse pressure observed during expiration and inspiration, respectively, is shown. There is no trend with time when mask pressure is zero (Figure 8). There is a progressive decrease in both the maximum and minimum pulse pressures during a breathing cycle at 60 mm mask pressure as the run progresses (Figure 7).
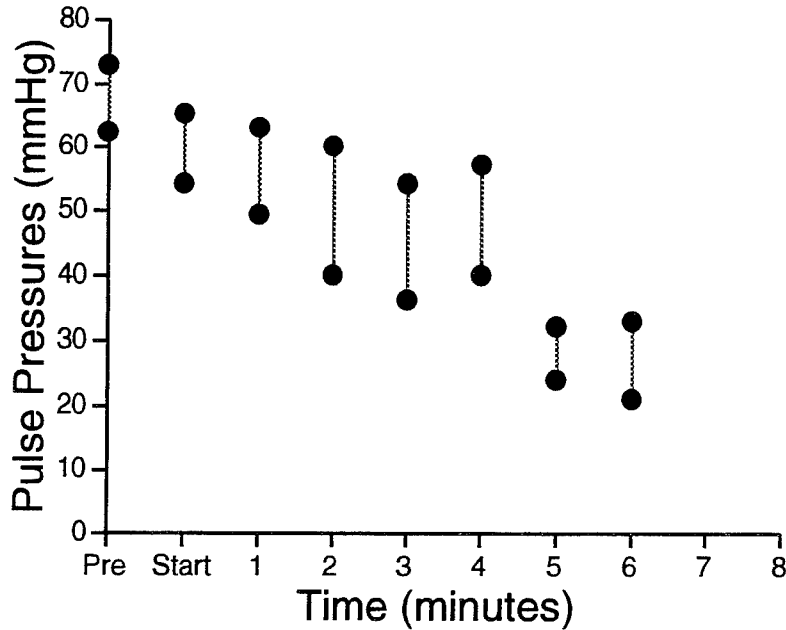
Figure 7: Respiratory variation in pulse pressure vs time (60/60).
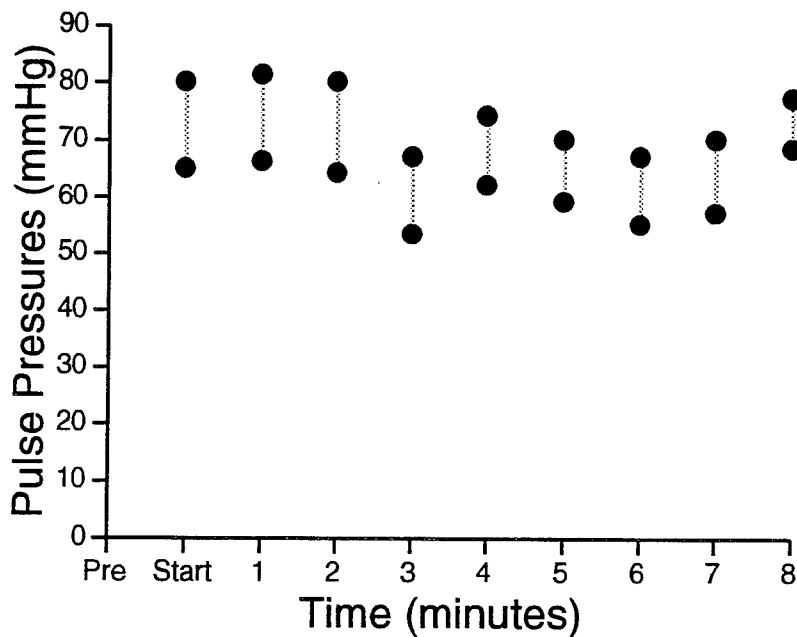
Figure 8: Respiratory variation in pulse pressure vs time (0/0).

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Increased mask pressure resulted in an increase in CVP (P = .0001) (Figure 9). This increase in CVP is in part due to the increased intrathoracic pressure. Pulmonary artery wedge pressure shows a similar trend and was generally around 5 mmHg higher than CVP. However, is it also possible that the catheter tip was lodged in a relatively under-perfused lung unit especially during high mask pressures, and therefore may not have represented left atrial pressure.
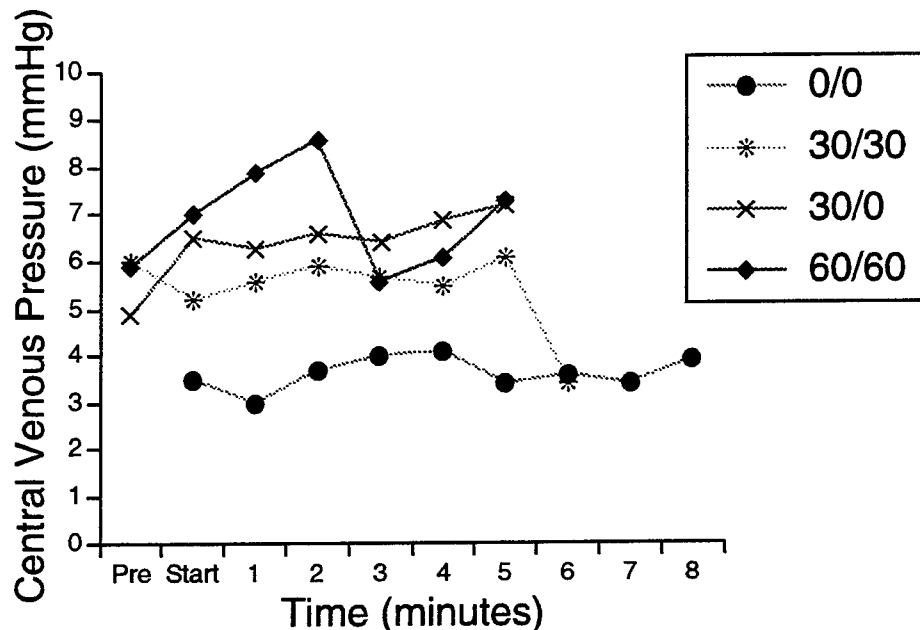
Figure 9: CVP vs time.

Increase in mask pressure resulted in reduced cardiac output (P = .03 by MIG and O_2 Fick) (Figure 10). Oxygen Fick cardiac outputs which were obtained during air breathing runs showed the same trend. There was more variability in thermodilution cardiac output because the value depended very highly upon the actual phase of respiration at which the injection of iced saline was made.
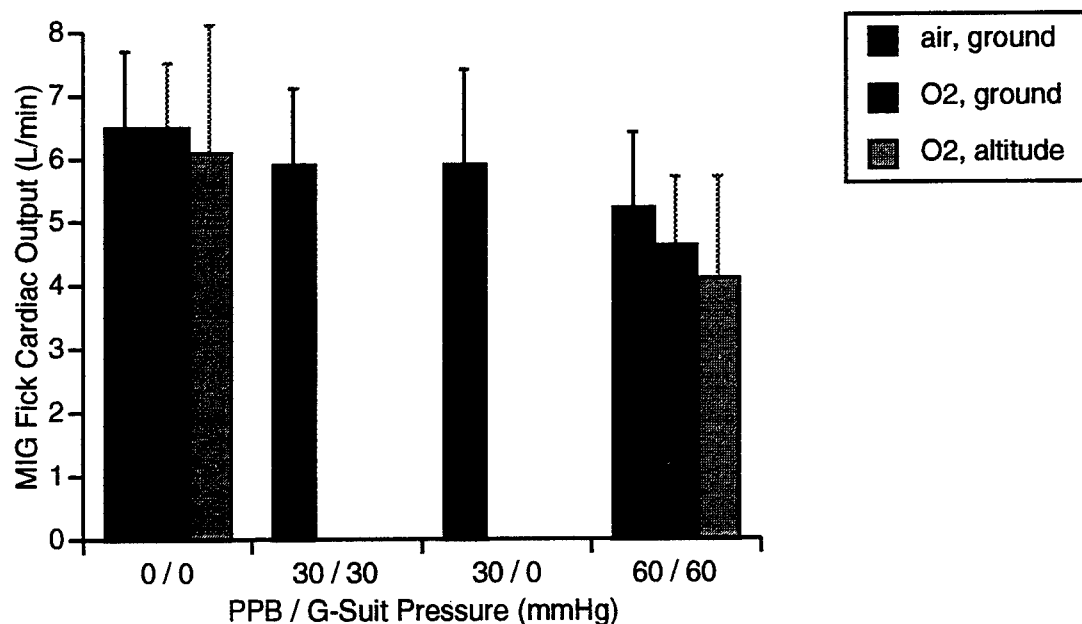
Figure 10: MIG Fick cardiac output vs condition.

Along with a reduction in cardiac output was an expected decrease in $S\bar{v}\,O_2$ (P < .0001) (Figure 11). $S\bar{v}$ O_2 was also affected by both altitude and breathing gas, since both indices affect the inspired PO_2 tension.
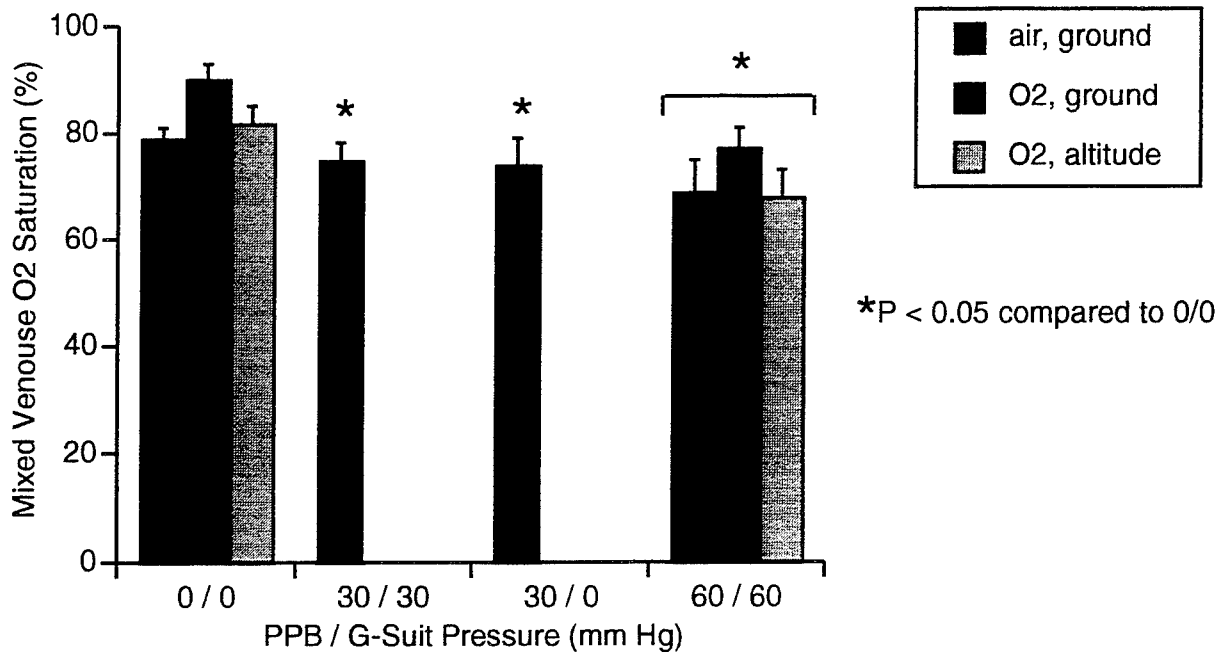
Figure 11: Mixed venous oxygen saturation vs condition.

Statistics

Matrices of probability values obtained from ANOVA are shown in Tables 1-3.

Duration of Pressure Breathing

Mean experimental run duration at zero mask pressure, indicating the time necessary to obtain all blood samples and hemodynamic measurements, was 12-13 minutes. Experimental duration was reduced to around 8 minutes at 30 mmHg and 4.5 minutes at 60 mmHg mask pressure. In part this shortened time was created by the experimenters, and was due to a reduced number of blood samples and shorter collection times, instituted because of subject stress. The collection of samples was adjusted in order to obtain at least one set of measurements in case the subject halted prematurely. Potential problems at 60 mmHg could often be predicted by observing the subject's ease at performing PPB of 30 mmHg in those instances when 30 mmHg runs preceded the 60 mmHg runs. Of 25 experimental runs begun at 60 mmHg mouthpiece pressure, 11 lasted 4 minutes or less, and two runs terminated at 4.7 and 4.9 minutes with loss of consciousness. Both episodes were at altitude. In one individual (JL) this coincided with a mean arterial pressure of 60 mmHg, loss of pulse pressure and bradycardia, suggestive of lack of brain perfusion. The other episode (TH) was accompanied by tetany, (hypocapnea arterial PCO_2 = 13.6 mmHg) and (respiratory alkalosis arterial pH = 7.74; $[H^+]$ = 18.2 nM). Two runs (subjects SA at altitude with PPB - Figure 27 and VW at ground level with 60 mmHg PPB) were terminated with marked tachypnea, consistent with respiratory muscle fatigue.

This study was not specifically designed to monitor maximum duration of PPB. It is possible that some individuals had difficulty completing the study because of inadequate motivation, a factor which could be overcome by training or, in actual combat, the knowledge of imminent death due to hypoxia. However, at least two foreshortened studies were due to physiological factors unlikely to be amenable to voluntary modification. All subjects felt that they were certainly close to their duration limits during 60 mmHg runs.

DISCUSSION

Gas exchange. Increasing mask pressure resulted in increase in $\dot{V}_A/\dot{Q}$ mismatching and a shift to lung units with higher $\dot{V}_A/\dot{Q}$. This partly confirmed the initial hypothesis and provided some explanation for the hyperventilation seen during positive pressure breathing. However, the hyperventilation that was observed was greater than required to maintain normocapnea. All subjects became hypocapneic, and one subject decreased his PCO_2 to 13.6 mmHg. The origin of this hyperventilation is not elucidated by this study. Our subjects were well-trained and it is unlikely that this represents anxiety. Subjects were unable to limit this hyperventilation even though they were all repeatedly instructed not to hyperventilate. It should be noted that because of the shift to high $\dot{V}_A/\dot{Q}$ ratios end-tidal PCO_2 would under-represent the arterial value. Future studies of gas exchange should therefore continue to include direct measurement of arterial gas values.

Ventilation to high $\dot{V}_A/\dot{Q}$ units tended to increase at altitude. Presumably this was an effect of the lower gas density at altitude, resulting in an alteration of breathing pattern compared to ground level.

After 45-90 minutes of 100% O_2 breathing there was a tendency toward an increase in low $\dot{V}_A/\dot{Q}$ units and shunt, presumably due to microatelectasis. There was a reduction in perfusion to these units during PPB, although this did not reach statistical significance. Although it is highly likely that PPB would minimize or reverse such changes, an experiment to prove this assertion would require longer duration of O_2 breathing.

Hemodynamics. The expected impairment of cardiac function was observed in these experiments, and limited PPB duration in at least one subject.

Limiting factors to the use of the COMBAT EDGE. Using the COMBAT EDGE in this configuration it is evident that subjects reach the limit of endurance within a few minutes. In addition to motivation, limiting factors include reduced cardiac output and cerebral blood flow, respiratory fatigue, pharyngeal muscle hypoperfusion and/or fatigue, hyperventilation and the possibility of pulmonary barotrauma (pnemothorax, mediastinal emphysema and arterial gas embolism).

There was significant inter-individual variability in tolerance of PPB. One of the major mechanisms of this intolerance is undoubtedly the reduction in cardiac output (most dramatically observed in subject JL - see Figure 12). This is presumably due to the reduced venous return caused by increased intrathoracic pressure, and possibly also the increased pulmonary vascular resistance associated with an elevated alveolar pressure. Figure 13 demonstrates that the transient augmentation of venous return by increasing G-suit pressure to 4:1 G-suit to mask ratio tends to normalize blood pressure. Perhaps some individuals are able to maintain a higher venous tone, either intrinsically or by active contraction of skeletal muscle.

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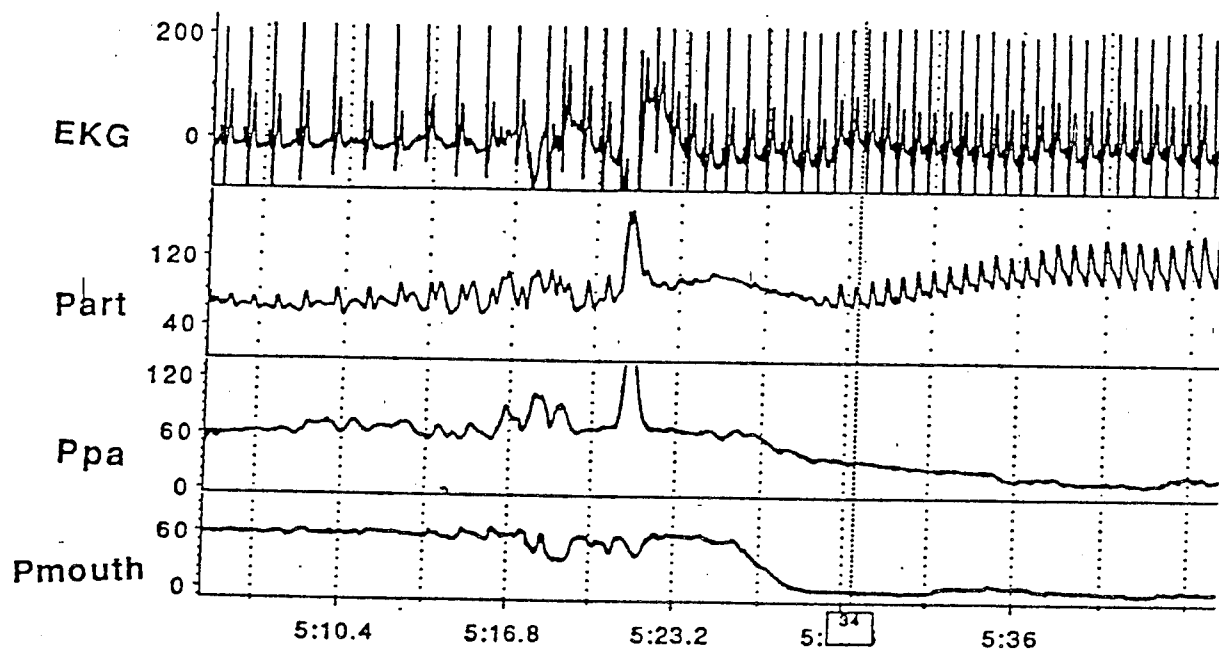
Figure 12. Loss of Consciousness.
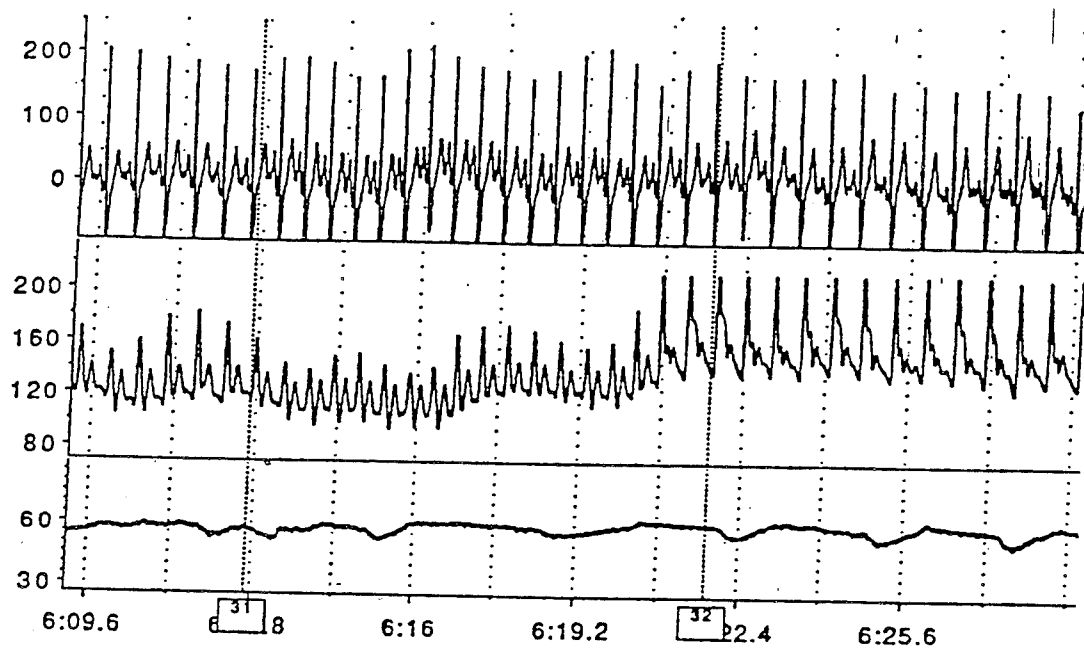
Figure 13. Increased G-Suit Pressure.

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It is noteworthy that mask leaks often resulted in transient increases in blood pressure. Such random leaks might be the reason that PPB using the COMBAT EDGE appears be better tolerated in actual use (Travis & Morgan, 1994) than in these laboratory experiments, in which every effort was made to maintain a good mask seal. Redesign of the system to provide a phasic pressure response might produce more reproducible, and safer, conditions for the pilot.

A 1:1 G-suit to mask pressure ratio was used in these studies as requested. 3:1 and 4:1 ratios improved subject performance and have unknown effects on gas exchange.

SUMMARY OF RESULTS

1. There was an almost 5-fold increase in minute ventilation at 60 mmHg mask pressure. The increase in minute ventilation was achieved by an increase in tidal volume but not of ventilatory frequency. The highest mask pressures resulted in a slight reduction in cardiac output.

2. PPB resulted in a shift of ventilation and perfusion to lung units at higher $\dot{V}_A/\dot{Q}$ s. At the extremes of the distribution (including dead space) these changes were relatively insignificant. The changes in ventilation/perfusion of the lung did not explain the extreme hyperpnea observed during PPB. The increase in ventilation of high $\dot{V}_A/\dot{Q}$ units would tend to increase the discrepancy between end-tidal and arterial PO_2. Therefore, assessment of arterial PCO_2 during high levels of PPB should ideally be obtained by direct measurement of arterial gas tensions.

3. The data support the notion that PPB reduces shunt and perfusion to low $\dot{V}_A/\dot{Q}$ lung units, though it appears that the periods of high oxygen breathing used in this study were not sufficiently long to result in significant increases in these variables and hence the result did not reach statistical significance.

4. While oxygen breathing resulted in a minor effect on some variables, there was a statistically significant effect of altitude exposure on several parameters. At 24,900 feet altitude heart rate was higher than at ground level. Arterial blood pressure was lower during PPB at altitude than at ground level. In addition, the respiratory effects on hemodynamics were less at altitude than at ground level, presumably because of lower gas density. The ability of the COMBAT EDGE regulator to maintain constant mask pressure was greater at altitude than at ground level. Minute ventilation tended to be higher at altitude than at ground level when mask pressure was elevated, though this did not reach statistical significance. Nevertheless, physiological testing of this apparatus at ground level is unlikely to predict fully its physiological effects under conditions of actual use.

5. Phasic swings in mask pressure seem to augment venous return and sustain mean arterial pressures in some people breathing with PPB at 1:1 G-suit to mask ratios. Increased G-suit pressures may augment blood return and maintain arterial pressures but have unknown effects on overall pulmonary gas exchange.

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Appendix

Summary of Blood Gases and Ventilation

Ground Level
Mean PB: 756 mmHg
Breathing gas: Air
Mask Pressure: 0 mmHg
Counterpressure: 0 mmHg
G-suit pressure: 0 mmHg

Subject	PvO2	PvCO2	pHv	PaO2	PaCO2	pHa	VO2	VCO2	SaO2	SvO2	VE	Vf	Vt	VD/VT*	VD/VT*	VD**	VD**
	mmHg	mmHg		mmHg	mmHg		ml/m	ml/m			l/m BTPS	br/m	l BTPS	(ambient)	(mask)	l (ambient)	l (mask)
RT	44	42.5	7.34	87	41.3	7.36	263	168	0.97	0.80	6.4	12.1	0.531	0.447	0.447	0.237	0.237
KK	37	39.1	7.40	109	34.4	7.43	270	220	0.99	0.72	8.1	9.1	0.886	0.305	0.305	0.270	0.270
FC	42	37.1	7.39	107	34.2	7.41	307	242	0.99	0.79	8.1	10.3	0.785	0.230	0.230	0.181	0.181
VW	42	36.8	7.39	98	36.2	7.42	209	158	0.99	0.80	6.4	11.9	0.541	0.394	0.394	0.213	0.213
WS	43	45.9	7.35	94	41.9	7.37	214	168	0.98	0.76	6.3	13.7	0.457	0.432	0.432	0.197	0.197
TH	45	41.7	7.39	108	36.5	7.40	250	215	0.99	0.83	7.8	10.5	0.742	0.336	0.336	0.249	0.249
JL	43	42.5	7.36	97	38.4	7.38	239	196	0.98	0.81	5.8	7.7	0.748	0.229	0.229	0.171	0.171
TLH	45	42.1	7.37	105	39.2	7.39	257	198	0.98	0.82	7.2	15.6	0.462	0.380	0.380	0.175	0.175
SA	41	41.3	7.37	99	38.3	7.39	328	252	0.99	0.78	7.7	10.9	0.706	0.249	0.249	0.176	0.176
TP	43	43.2	7.35	110	38.0	7.38	200	171	0.99	0.78	7.3	20.0	0.365	0.456	0.456	0.166	0.166
MEAN	43	41.2	7.37	101	37.8	7.39	254	199	0.98	0.79	7.1	12.2	0.622	0.346	0.346	0.204	0.204
STD DEV	2	2.8	0.02	8	2.6	0.02	41	33	0.01	0.03	0.8	3.5	0.172	0.089	0.089	0.037	0.037

VD/DT, calculated using the Enghoff modification of the Bohr equation.
*VD/VT has been calculated using two different values for Pb: ambient (chamber) pressure and mask pressure.
**Both volumes are reported in liters BTPS, using ambient (chamber) pressure.

Summary of Blood Gases and Ventilation (cont'd)

Ground Level
Mean PB: 756 mmHg
Breathing gas: Air
Mask Pressure: 30 mmHg
Counterpressure: 30 mmHg
G-suit pressure   30 mmHg

Subject	PvO2	PvCO2	pHv	PaO2	PaCO2	pHa	VO2	VCO2	SaO2	SvO2	VE	Vf	Vt	VD/VT*	VD/VT*	VD**	VD**
	mmHg	mmHg		mmHg	mmHg		ml/m	ml/m			l/m BTPS	br/m	l BTPS	(ambient)	(mask)	l (ambient)	l (mask)
RT	35	30.5	7.42	129	27.3	7.49	267	272	0.99	0.74	16.4	8.3	1.972	0.461	0.438	0.909	0.864
KK	31	32.0	7.44	134	27.3	7.52	350	380	0.99	0.67	24.7	20.2	1.222	0.488	0.466	0.596	0.569
FG	35	34.5	7.46	127	25.8	7.53	268	337	1.00	0.71	17.3	5.5	3.151	0.329	0.301	1.037	0.948
VW	37	30.4	7.51	146	21.3	7.61	379	586	1.00	0.78	45.4	30.4	1.493	0.450	0.426	0.671	0.636
WS	36	28.4	7.51	144	20.5	7.63	297	474	1.00	0.74	32.8	13.7	2.391	0.363	0.335	0.867	0.801
TH	33	26.2	7.57	150	17.4	7.68	318	582	1.00	0.78	44.8	14.8	3.028	0.331	0.303	1.001	0.918
JL	36	37.4	7.41	118	31.4	7.46	221	260	0.99	0.74	13.8	8.4	1.637	0.467	0.444	0.764	0.727
TLH	38	28.4	7.51	149	22.3	7.59	277	427	1.00	0.81	25.2	9.7	2.595	0.318	0.289	0.825	0.750
SA	34	32.6	7.46	124	27.8	7.51	318	371	0.99	0.73	18.3	6.7	2.725	0.355	0.327	0.966	0.891
TP	36	30.5	7.50	131	24.3	7.57	153	315	0.99	0.76	18.9	12.4	1.521	0.386	0.360	0.587	0.548
MEAN	35	31.1	7.48	135	24.5	7.56	285	400	1.00	0.75	25.7	13.0	2.174	0.395	0.369	0.822	0.920
STD DEV	2	3.2	0.05	11	4.2	0.07	65	117	0.00	0.04	11.6	7.5	0.694	0.065	0.068	0.164	0.144

VD/DT, calculated using the Enghoff modification of the Bohr equation.
*VD/VT has been calculated using two different values for Pb: ambient (chamber) pressure and mask pressure.
**Both volumes are reported in liters BTPS, using ambient (chamber) pressure.

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Summary of Blood Gases and Ventilation (cont'd)

Ground Level

Mean PB:	756 mmHg
Breathing gas:	Air
Mask Pressure:	30 mmHg
Counterpressure:	0 mmHg
G-suit pressure	0 mmHg

Subject	PvO2 mmHg	PvCO2 mmHg	pHv	PaO2 mmHg	PaCO2 mmHg	pHa	VO2 ml/m	VCO2 ml/m	SaO2	SvO2	VE l/m BTPS	Vf br/m	Vt l BTPS	VD/VT* (ambient)	VD/VT* (mask)	VD** l (ambient)	VD** l (mask)
RT	31	27.8	7.47	127	24.9	7.53	311	329	0.99	0.70	20.3	9.1	2.227	0.423	0.399	0.942	0.889
KK	30	33.6	7.48	134	24.5	7.55	280	340	1.00	0.65	23.2	16.9	1.373	0.456	0.433	0.626	0.594
FC	33	29.3	7.46	135	24.0	7.55	299	383	1.00	0.70	21.4	6.2	3.445	0.335	0.307	1.152	1.058
VW	45	27.0	7.54	145	18.6	7.67	291	771	1.00	0.89	58.9	23.1	2.550	0.362	0.335	0.923	0.854
WS	34	27.1	7.56	147	17.5	7.68	292	666	1.00	0.75	50.5	15.8	3.194	0.316	0.287	1.011	0.917
TH	31	26.8	7.57	149	17.4	7.68	285	568	1.00	0.74	49.8	18.8	2.648	0.410	0.385	1.086	1.019
JL	33	35.8	7.43	127	26.9	7.51	236	311	0.99	0.70	17.9	8.1	2.211	0.427	0.403	0.945	0.891
TLH	39	32.1	7.47	129	28.4	7.51	274	370	1.00	0.80	20.0	11.1	1.799	0.415	0.390	0.747	0.702
SA	32	30.8	7.48	130	23.6	7.56	293	403	1.00	0.70	22.6	7.7	2.939	0.332	0.304	0.977	0.893
TP	36	31.0	7.49	146	18.9	7.61	278	569	1.00	0.77	37.7	15.8	2.385	0.285	0.254	0.680	0.606
MEAN	34	30.1	7.49	137	22.5	7.58	284	471	1.00	0.74	32.2	13.3	2.477	0.376	0.350	0.909	0.930
STD DEV	5	3.1	0.05	9	4.0	0.07	20	161	0.00	0.07	15.5	5.6	0.625	0.057	0.060	0.172	0.159

VD/DT, calculated using the Enghoff modification of the Bohr equation.
*VD/VT has been calculated using two different values for Pb: ambient (chamber) pressure and mask pressure.
**Both volumes are reported in liters BTPS, using ambient (chamber) pressure.

Ground Level

Mean PB:	756 mmHg
Breathing gas:	Air
Mask Pressure:	60 mmHg
Counterpressure:	60 mmHg
G-suit pressure:	60 mmHg

Subject	PvO2 mmHg	PvCO2 mmHg	pHv	PaO2 mmHg	PaCO2 mmHg	pHa	VO2 ml/m	VCO2 ml/m	SaO2	SvO2	VE l/m BTPS	Vf br/m	Vt l BTPS	VD/VT* (ambient)	VD/VT* (mask)	VD** l (ambient)	VD** l (mask)
RT	27	32.2	7.47	134	23.3	7.55	306	333	1.00	0.60	23.1	9.8	2.352	0.446	0.400	1.050	0.941
KK	28	37.9	7.42	119	30.9	7.47	340	280	1.00	0.55	21.9	30.9	0.707	0.629	0.598	0.445	0.423
FC	33	17.5	7.46	138	21.9	7.57	350	451	1.00	0.71	23.0	8.1	2.838	0.209	0.143	0.592	0.406
VW	30	24.1	7.58	156	18.1	7.66	480	803	1.00	0.70	47.4			0.402	0.351		
WS	37	30.0	7.48	143	20.2	7.64	180	534	1.00	0.77	40.8	20.5	1.992	0.412	0.362	0.821	0.721
TH	31	27.3	7.56	152	15.6	7.71	338	722	1.00	0.73	62.6	20.4	3.069	0.336	0.280	1.030	0.859
JL	31	35.7	7.43	135	26.1	7.52	169	270	0.99	0.65	16.2	9.4	1.721	0.432	0.384	0.744	0.661
TLH	36	31.2	7.48	148	22.4	7.58	204	608	1.00	0.77	35.9	10.4	3.456	0.323	0.266	1.115	0.919
SA				132	20.3	7.62	288	473	1.00		32.6	10.9	2.987	0.362	0.308	1.082	0.920
TP	33	32.1	7.48	146	21.4	7.61	232	377	1.00	0.72	23.7	13.8	1.714	0.334	0.277	0.573	0.475
MEAN	32	29.8	7.48	140	22.0	7.59	289	485	1.00	0.69	31.1	18.2	2.315	0.389	0.337	0.828	0.849
STD DEV	3	6.2	0.05	11	4.2	0.07	96	182	-0.00	0.07	14.2	12.5	0.866	0.109	0.118	0.253	0.222

VD/DT, calculated using the Enghoff modification of the Bohr equation.
*VD/VT has been calculated using two different values for Pb: ambient (chamber) pressure and mask pressure.
**Both volumes are reported in liters BTPS, using ambient (chamber) pressure.

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Summary of Blood Gases and Ventilation (cont'd).

Ground Level

Mean PB:	756 mmHg
Breathing gas:	100% oxygen
Mask Pressure:	0 mmHg
Counterpressure:	0 mmHg
G-suit pressure	0 mmHg –

Subject	PvO2 mmHg	PvCO2 mmHg	pHv	PaO2 mmHg	PaCO2 mmHg	pHa	VO2† ml/m	VCO2 ml/m	SaO2	SvO2	VE l/m BTPS	Vf br/m	Vt l BTPS	VD/VT* (ambient)	VD/VT* (mask)	VD** l (ambient)	VD** l (mask)
RT	63	44.7	7.34	587	37.6	7.37		200	1.00	0.93	7.8	15.1	0.517	0.409	0.409	0.211	0.211
KK	46	42.3	7.37	574	35.8	7.42		212	1.00	0.83	8.7	14.4	0.603	0.411	0.411	0.248	0.248
PC	54	38.0	7.41	626	33.7	7.45		255	1.00	0.89	8.7	5.9	1.480	0.250	0.250	0.370	0.370
VW	55	40.8	7.40	563	35.3	7.43		229	1.00	0.90	9.3	16.0	0.583	0.398	0.398	0.232	0.232
WS	53	42.6	7.37	585	38.3	7.40		178	1.00	0.87	7.4	15.4	0.479	0.455	0.455	0.218	0.218
TH	65	41.3	7.39	573	34.0	7.43		179	1.00	0.95	8.6	10.5	0.632	0.315	0.315	0.199	0.199
JL	55	41.9	7.36	579	37.1	7.39		204	1.00	0.90	6.2	8.3	0.747	0.235	0.235	0.175	0.175
TLH	61	41.7	7.37	590	36.5	7.42		220	1.00	0.93	7.9	15.6	0.506	0.342	0.342	0.173	0.173
SA	49	37.7	7.39	560	34.0	7.43		238	1.00	0.88	8.4	11.1	0.759	0.279	0.279	0.212	0.212
TP	54	42.0	7.36	555	35.8	7.41		196	1.00	-0.88	8.0	14.8	0.543	0.410	0.410	0.222	0.222
MEAN	56	41.3	7.38	579	35.8	7.41		211	1.00	0.90	7.9	12.7	0.685	0.350	0.350	0.226	0.226
STD DEV	6	2.1	0.02	20	1.6	0.02		25	0.00	0.04	1.0	3.5	0.295	0.077	0.077	0.056	0.056

VD/DT, calculated using the Enghoff modification of the Bohr equation.

*VD/VT has been calculated using two different values for Pb: ambient (chamber) pressure and mask pressure.

**Both volumes are reported in liters BTPS, using ambient (chamber) pressure.

†VO2 could not be calculated during 100% O2 breathing because of the open circuit technique used in this experiment.

Summary of Blood Gases and Ventilation (cont'd)

Ground Level

Mean PB:	756 mmHg
Breathing gas:	100% oxygen
Mask Pressure:	60 mmHg
Counterpressure:	60 mmHg
G-suit pressure	60 mmHg

Subject	PvO2 mmHg	PvCO2 mmHg	pHv	PaO2 mmHg	PaCO2 mmHg	pHa	VO2† ml/m	5 ml/m	SaO2	SvO2	VE l/m BTPS	Vf br/m	Vt l BTPS	VD/VT* (ambient)	VD/VT* (mask)	VD** l (ambient)	VD** l (mask)
RT	34	35.2	7.44	664	24.1	7.55		285	1.00	0.72	18.7	12.5	1.495	0.453	0.407	0.678	0.609
KK	36	34.2	7.44	820	27.1	7.53		402	1.00	0.74	32.6	35.7	0.913	0.607	0.574-	0.554	0.524
FC																	
VW																	
WS	36	28.5	7.52	585	16.7	7.69		615	1.00	0.76	44.7	21.4	2.087	0.362	0.308	0.756	0.643
TH																	
JL	37	35.1	7.42	630	26.2	7.52		279	1.00	0.74	16.9	10.5	1.612	0.461	0.415	0.743	0.669
TLH	41	29.5	7.49	696	21.7	7.60		456	1.00	0.85	27.0	12.0	2.248	0.327	0.271	0.736	0.609
SA	32	27.0	7.52	803	19.1	7.63		396	1.00	0.74	30.5	10.1	3.017	0.411	0.361	1.240	1.089
TP	41	32.1	7.48	590	20.6	7.62		359	1.00	0.82	21.8	13.5	1.618	0.308	0.248	0.499	0.401
MEAN	37	31.7	7.47	627	22.2	7.59		399	1.00	0.77	27.5	16.5	1.856	0.418	0.369	0.744	0.872
STD DEV	3	3.4	0.04	41	3.8	0.06		115	0.00	0.05	9.6	9.3	0.670	0.102	0.111	0.240	0.214

VD/DT, calculated using the Enghoff modification of the Bohr equation.
*VD/VT has been calculated using two different values for Pb: ambient (chamber) pressure and mask pressure.
**Both volumes are reported in liters BTPS, using ambient (chamber) pressure.
†VO2 could not be calculated during 100% O2 breathing because of the open circuit technique used in this experiment.

Summary of Blood Gases and Ventilation (cont'd)

24,900 ft
Mean PB: 283 mmHg
Breathing gas: 100% oxygen
Mask Pressure: 0 mmHg
Counterpressure: 0 mmHg
G-suit pressure 0 mmHg

Subject	PvO2	PvCO2	pHv	PaO2	PaCO2	pHa	VO2†	VCO2	SaO2	SvO2	VE	Vf	Vt	VD/VT*	VD/VT*	VD**	VD**
	mmHg	mmHg		mmHg	mmHg		ml/m	ml/m			l/m BTPS	br/m	l BTPS	(ambient)	(mask)	l (ambient)	l (mask)
RT	43	41.0	7.35	168	38.7	7.37		192	0.99	0.79	8.3	16.9	0.489	0.478	0.478	0.234	0.234
KK																	
FC	47	38.7	7.41	181	36.7	7.43		224	1.00	0.80	9.0	-8.2	1.101	0.418	0.418	0.461	0.461
VW	47	40.5	7.40	191	35.8	7.41		230	1.00	0.83	9.6	13.3	0.718	0.420	0.420	0.302	0.302
WS	42	39.8	7.39	182	35.7	7.41		188	1.00	0.77	7.7	14.5	0.534	0.407	0.407	0.217	0.217
TH	52	43.3	7.37	198	38.5	7.38		138	1.00	0.88	8.6	13.3	0.644	0.636	0.636	0.409	0.409
JL	47	41.2	7.36	182	28.7	7.37		153	1.00	0.84	6.1	10.6	0.576	0.242	0.242	0.140	0.140
TLH	48	40.7	7.37	193	37.7	7.39		165	1.00	0.85	7.1	16.9	0.421	0.467	0.467	0.197	0.197
SA	41	38.0	7.37	184	38.0	7.37		225	0.99	0.78	9.1	9.5	0.953	0.438	0.438	0.418	0.418
TP																	
MEAN	46	40.4	7.38	185	36.2	7.39		189	0.99	0.82	8.2	12.9	0.679	0.439	0.439	0.297	0.297
STD DEV	4	1.6	0.02	9	3.2	0.02		35	0.00	0.04	1.1	3.2	0.236	0.108	0.108	0.119	0.119

VD/DT, calculated using the Enghoff modification of the Bohr equation.
*VD/VT has been calculated using two different values for Pb: ambient (chamber) pressure and mask pressure.
**Both volumes are reported in liters BTPS, using ambient (chamber) pressure.
†VO2 could not be calculated during 100% O2 breathing because of the open circuit technique used in this experiment.

Summary of Blood Gases and Ventilation (cont'd)

24,900 ft
Mean PB: 283 mmHg
Breathing gas: 100% oxygen
Mask Pressure: 60 mmHg
Counterpressure: 60 mmHg
G-suit pressure: 60 mmHg

Subject	PvO2 mmHg	PvCO2 mmHg	pHv	PaO2 mmHg	PaCO2 mmHg	pHa	VO2† ml/m	VCO2 ml/m	SaO2	SvO2	VE l/m BTPS	Vf br/m	Vt l BTPS	VD/VT* (ambient)	VD/VT* (mask)	VD** l (ambient)	VD** l (mask)
RT	36	32.8	7.47	206	24.5	7.53		107	1.00	0.75	9.6	16.7	0.572	0.594	0.490	0.340	0.280
KK																	
FC	39	34.4	7.46	270	23.9	7.56		400	1.00	0.67	25.2	12.1	2.082	0.425	0.279	0.885	0.581
VW																	
WS	29	35.9	7.46	247	17.0	7.67		467	1.00	0.58		18.0		0.471	0.337		
TH	31	23.7	7.59	256	13.6	7.74		642	1.00	0.75	81.0	22.9	3.538	0.494	0.365	1.747	1.292
JL	28	34.3	7.44	238	23.6	7.55		291	1.00	0.60	22.3	10.0	2.234	0.525	0.405	1.174	0.905
TLH	35	30.7	7.48	255	19.9	7.62		470	1.00	0.76	31.6	13.3	2.374	0.349	0.184	0.830	0.437
SA	30	31.9	7.48	246	20.8	7.60		496	1.00	0.67	36.6	19.4	1.884	0.436	0.292	0.821	0.550
TP	31	31.2	7.47	244	14.8	7.70		638	1.00	0.67	68.3	28.8	2.373	0.457	0.319	1.085	0.757
MEAN	32	31.9	7.48	245	19.8	7.62		439	1.00	0.68	39.2	17.7	2.151	0.469	0.334	0.983	0.698
STD DEV	4	3.7	0.05	19	4.2	0.08		177	0.00	0.07	25.9	6.1	0.876	0.073	0.091	0.429	0.336

VD/DT, calculated using the Enghoff modification of the Bohr equation.
*VD/VT has been calculated using two different values for Pb: ambient (chamber) pressure and mask pressure.
**Both volumes are reported in liters BTPS, using ambient (chamber) pressure.
†VO2 could not be calculated during 100% O2 breathing because of the open circuit technique used in this experiment.

Cardiac Output, Heart Rate, CVP, Mean Arterial and PA Pressures

Ground Level

Mean PB:	756 mmHg
Breathing gas:	Air
Mask Pressure:	0 mmHg
Counterpressure:	0 mmHg
G-suit pressure:	0 mmHg

Subject	Cardiac Output			HR Start	HR Mid	HR End	Part Start	Part Mid	Part End	Ppa Start	Ppa Mid	Ppa End	CVP Start	CVP Mid	CVP End
	O2	MIG	TD												
	l/m	l/m	l/m												
RT	7.3	5.1		•	56	58	•	77	74	•	0.0	-2.0	•	-4.1	-4.2
KK	6.4	4.5	5.0	66	65	60	96	94	93	9.7	•	10.2	2.2	3.0	2.4
FC	8.5	8.0	6.6	65	64	•	•	90	89	•	8.6	7.2	•	0.2	-0.4
VW	5.8	5.1	7.1	82	78	75	107	100	95	4.2	7.8	•	-4.6	-1.7	-1.8
WS	5.7	5.8	5.3	59	63	64	98	104	97	6.2	•	7.5	-2.4	-2.0	-2.8
TH	8.7	7.3	6.3	85	80	90	74	76	74	4.0	8.0	4.5	-4.2	-5.0	-5.5
JL	7.7	7.2	5.7	86	75	76	90	87	90	0.5	4.0	1.9	•	-7.4	-7.3
TLH	7.9	8.2	6.5	87	81	79	105	100	96	12.1	12.2	10.2	2.6	1.8	1.9
SA	9.4	9.0	8.4	77	90	79	99	102	94	11.0	13.4	10.5	3.6	2.8	3.9
TP	4.9	4.4	4.7	74	65	67	85	87	88	7.1	8.7	6.6	-3.5	-3.4	-3.7
MEAN	7.22	6.48	6.16	75.67	71.70	72.00	94.25	91.70	89.00	6.85	7.84	6.29	-0.90	-1.58	-1.75
STD DEV	1.47	1.69	1.16	10.32	10.60	10.46	10.90	10.06	8.45	3.94	4.27	4.21	3.55	3.50	3.65

O2 Fick, calculated using the Fick principle from oxygen consumption and the a-v oxygen content difference; MIG Fick, calculated using the Fick principle from the four least soluble inert gases; TD, determined by thermodilution.

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Cardiac Output, Heart Rate, CVP, Mean Arterial and PA Pressures

Ground Level

Mean PB:	756 mmHg
Breathing gas:	Air
Mask Pressure:	30 mmHg
Counterpressure:	30 mmHg
G-suit pressure:	30 mmHg

Subject	Cardiac Output			HR Start	HR Mid	HR End	Part Start	Part Mid	Part End	Ppa Start	Ppa Mid	Ppa End	CVP Start	CVP Mid	CVP End
	O2 l/m	MIG l/m	TD l/m												
RT	5.2	3.7		73	75	72	87	87	95	18.9	18.7	14.7	19.1	18.8	18.1
KK	7.0	4.3	4.1	66	73	78	122	129	129	35.1	35.2	37.7	34.9	37.7	37.2
FC	4.9	4.9	5.7	84	82	95	112	102	113	35	34.5	37.2	23.5	22.5	24.8
VW	8.9	8.1	6.9	98	119	112	115	125	125	31.6	34.6	29.5	21.1	-23.7	23.1
WS	6.6	5.9	6.0	92	104	102	8-1	108	107	28.3	30.1	28.9	21	26.2	22.2
TH	7.1	8.1	6.8	111	126	109	87	96	101	27.4	27.8	30.9	15.9	16.5	18.8
JL	4.7	4.8	5.6	100	99	91	123	123	121	32.3	35.5	33.4	.	.	28.2
TLH	7.1	7.3	5.6	112	116	113	119	126	123	35.2	39.3	35.4	25	24.1	23.7
SA	6.7	7.0	7.2	80	92	85	112	114	109	32	36	33.6	23.3	24	29.6
TP	3.5	4.8	4.8	82	94	87	103	99	98	31.7	38.3	23.8	23.8	23.2	23
MEAN	6.18	5.89	5.83	89.80	98.00	94.40	106.10	110.90	112.10	30.75	33.00	30.51	23.07	24.08	24.87
STD DEV	1.57	1.63	1.00	15.48	18.40	14.35	15.72	14.65	12.02	4.94	6.08	6.97	5.24	5.90	5.61

O2 Fick, calculated using the Fick principle from oxygen consumption and the a-v oxygen content difference; MIG Fick, calculated using the Fick principle from the four least soluble inert gases; TD, determined by thermodilution.

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Cardiac Output, Heart Rate, CVP, Mean Arterial and PA Pressures

Ground Level
Mean PB: 756 mmHg
Breathing gas: Air
Mask Pressure: 30 mmHg
Counterpressure: 0 mmHg
G-suit pressure: 0 mmHg

Subject	Cardiac Output			HR Start	HR Mid	HR End	Part Start	Part Mid	Part End	Ppa Start	Ppa Mid	Ppa End	CVP Start	CVP Mid	CVP End
	O2	MIG	TD												
	l/m	l/m	l/m												
RT	4.8	5.0		71	81	92	94	94	83	25.3	25.9	28.7	18	19.2	21.8
KK	4.9	3.8	4.8	70	84	82	114	121	120	42.9	44.3	47.6	29.8	30.2	30.6
FC	5.5	3.9	8.6	89	91	86	107	107	108	30.8	31.2	28.9	30	29.8	30.4
VW	12.2	10.6	10.2	102	128	122	106	110	121	27.7	29.6	34.4	13.1	19.4	18.3
WS	6.8	7.3	6.0	80	114	105	92	98	99	28.6	32.1	24.3	16	16.1	16
TH	5.3	6.2	6.2	94	103	148	86	91	94	28.1	29.4	31.7	13.5	12.9	13.4
JL	4.2	4.0	2.7	112	106	108	111	110	111	32.5	35.2	34.3	•	28.4	27.8
TLH	7.0	7.2	6.4	117	109	111	116	115	102	32.8	31.1	31.4	21.7	14.9	12.7
SA	5.4	5.7	5.9	102	97	95	104	102	101	29.6	34.6	31.9	21.5	21.5	21.6
TP	5.8	5.3	5.3	96	103	104	94	98	97	28.1	30.2	31.9	15	15.5	16.3
MEAN	6.2	5.9	6.2	93.3	101.6	105.3	102.4	104.6	103.6	30.64	32.36	32.51	19.84	20.79	20.89
STD DEV	2.3	2.1	2.1	16.01	14.13	19.27	10.27	9.62	11.72	4.87	4.9601	6.07	6.48	6.49	6.74

O2 Fick, calculated using the Fick principle from oxygen consumption and the a-v oxygen content difference; MIG Fick, calculated using the Fick principle from the four least soluble inert gases; TD, determined by thermodilution.

Cardiac Output, Heart Rate, CVP, Mean Arterial and PA Pressures

Ground Level
Mean PB: 756 mmHg
Breathing gas: Air
Mask Pressure: 60 mmHg
Counterpressure: 60 mmHg
G-suit pressure: 60 mmHg

Subject	Cardiac Output			HR Start	HR Mid	HR End	Part Start	Part Mid	Part End	Ppa Start	Ppa Mid	Ppa End	CVP Start	CVP Mid	CVP End
	O2	MIG	TD												
	l/m	l/m	l/m												
RT	3.7	4.1		78	88	105	112	114	104	50.4	70.4	51.9	44.4	49.2	45.8
KK	4.7	4.2	4.7	84	89	90	118	131	133	52.3	48.3	56.7	35.1	38.8	36.9
FC	6.3		5.3	112	96	112	133	147	146	59.1	64	77.8	56.2	56.4	55.4
VW	8.2		8.1	119	119	•	123	134	•	52.5	52.9	•	44.2	42.6	•
WS	4.6	6.0	7.2	110	109	113	86	101	109	54.5	46	50.8	45.2	34.3	44.3
TH	6.3	7.0		142	130	130	98	111	108	49.6	48.4	53.9	35.7	32.9	36.7
JL	2.8	3.6	3.3	106	123	109	135	129	106	60	62.3	60.6	•	57	•
TLH	4.4	7.0	8.0	131	171	140	136	138	138	60.9	61.6	52.5	51.8	47.3	35.2
SA	1.7	6.6	6.7	104	115	116	115	120	122	56.1	59.5	58.3	51.3	51.9	50.9
TP	4.2	3.6	4.1	94	129	131	117	114	116	56	51.5	57	47.2	44.7	45.8
MEAN	4.7	5.2	5.9	108	116.9	116.2	117.3	123.9	120.222	55.14	56.49	57.722	45.68	45.51	43.88
STD DEV	1.9	1.5	1.8	19.7709	24.5	15.28	16.08	14.19	15.43	3.98	8.16	8.19	7.05	8.48	7.22

O2 Fick, calculated using the Fick principle from oxygen consumption and the a-v oxygen content difference; MIG Fick, calculated using the Fick principle from the four least soluble inert gases; TD, determined by thermodilution.

Cardiac Output, Heart Rate, CVP, Mean Arterial and PA Pressures

Subject	Cardiac Output			HR Start	HR Mid	HR End	Part Start	Part Mid	Part End	Ppa Start	Ppa Mid	Ppa End	CVP Start	CVP Mid	CVP End
	O2 l/m	MG l/m	TD l/m												
RT	8.9			71	63	63	83.4	78.7	81.6	7.5	•	•	0.9	1.3	1.2
KK	5.3		7.0	58	67	65	96.9	101.8	100.2	7.4	•	5.2	0.6	0.6	1
FC	6.7		7.1	72	-64	65	87.6	89.1	85.8	6.9	•	•	-0.4	-0.7	-0.8
VW	6.6		6.2	69	77	68	96.2	96.8	97.8	•	6.7	6.7	-4.2	-4.9	•
WS	5.2		4.5	59	56	57	100.7	95.8	99.6	4.1	4.9	•	-3.8	-4.2	-5.2
TH	8.1		6.6	79	81	81	81.8	82.1	79.7	•	•	0.3	-3	-3.5	-3.5
JL	6.6		6.4	71	73	73	96.9	97	98.9	•	9.8	10.4	0.5	•	0.3
TLH	6.3		6.2	82	78	84	91.9	92	91.6	7.6	8.5	7.5	-3.5	-3.8	-3.5
SA	7.3		6.1	70	81	65	95	95.6	97.3	7.8	10.3	7.4	0.9	1.5	0.5
TP	4.1		4.6	70	71	77	85.7	85.4	83.8	4.6	6.7	5	-3.8	-3.7	-4.2
MEAN	6.5		6.1	70.1	71.1	69.8	91.61	91.43	91.63	6.56	7.82	6.07	-1.58	-1.9333	-1.58
STD DEV	1.4		0.9	7.43	8.45	8.64	6.55	7.42	8.15	1.54	2.08	3.11	2.24	2.58	2.50

O2 Fick, calculated using the Fick principle from oxygen consumption and the a-v oxygen content difference; MIG Fick, calculated using the Fick principle from the four least soluble inert gases; TD, determined by thermodilution.

Ground Level

Mean PB:	756 mmHg
Breathing gas:	100% oxygen
Mask Pressure:	60 mmHg
Counterpressure:	60 mmHg
G-suit pressure:	60 mmHg

Subject	Cardiac Output			HR Start	HR Mid	HR End	Part Start	Part Mid	Part End	Ppa Start	Ppa Mid	Ppa End	CVP Start	CVP Mid	CVP End
	O2	MIG	TD												
	l/m	l/m	l/m												
RT		2.9		77	81	80	110	127.1	126.1	44.9	49.4	47.2	37.6	42.2	44.5
KK		5.1	5.9	86	85	86	123.7	132.2	132.5	57.8	55.3		45.7	44.9	44.5
FC				•	•	•	•	•	—	•	•	•	•	•	•
VW				•	•	•	•	•		•	•	•	•	•	•
WS		5.4	6.5	100	113	96	89.1	120.7	124	54.3	•	•	41.8	39.2	44.3
TH				•	•	•	•	•	•	•	•	•	•	•	•
JL		2.9		113	120	127	141.1	141.9	142.9	61.7	65.8	65.3	•	59.9	•
TLH		6.1	7.4	109	126	127	139	138.1	138.2	58.5	57.6	58.1	42.3	35.2	34.4
SA		5.6	5.0	91	97	82	119.8	132.7	132.5	57.6	•	59.2	49.6	51.9	52.5
TP		4.3	4.3	101	122	117	117.2	117.1	118.4	57.6	40.2	56.9	48.4	43.9	46.2
MEAN		4.6	5.5	96.71	106.3	102.1	119.99	129.971	130.657	56.06	53.66	57.34	44.23	45.314	44.4
STD DEV		1.3	1.4	12.7895	18.47	21.02	17.71	8.95	8.45	5.37	9.55	6.53	4.52	8.24	5.81

O2 Fick, calculated using the Fick principle from oxygen consumption and the a-v oxygen content difference; MIG Fick, calculated using the Fick principle from the four least soluble inert gases; TD, determined by thermodilution.

Cardiac Output, Heart Rate, CVP, Mean Arterial and PA Pressures

24,900 ft
Mean PB: 283 mmHg
Breathing gas: 100% oxygen
Mask Pressure: 0 mmHg
Counterpressure: 0 mmHg
G-suit pressure: 0 mmHg

Subject	Cardiac Output			HR Start	HR Mid	HR End	Part Start	Part Mid	Part End	Ppa Start	Ppa Mid	Ppa End	CVP Start	CVP Mid	CVP End
	O2 l/m	MIG l/m	TD l/m												
RT	5.7			61	60	66	83.3	82.7	84.8	·	·	·	-0.9	-0.4	-0.2
KK				·	·	·	·	·	·	·	·	·	·	·	·
FC		0.8	5.9	65	74	70	96.5	99	97	8.9	9.1	8.2	-3.5	3.3	4.9
VW		8.9	7.7	75	74	83	100	108.4	92	9	·	9.8	0.4	-3.2	-0.1
WS		5.4	5.3	69	67	61	96.4	98.5	98.1	2.8	3.7	·	0.9	2.4	2.4
TH		6.1	6.3	74	80	81	87.7	86.4	83.2	5.6	·	8.1	0.4	-0.2	1.2
JL		6.8	6.1	89	81	82	90.9	87.8	92.7	7.8	8.9	10.6	5.5	4.9	9.7
TLH		6.2	6.3	90	89	86	93.9	94.6	94.1	7.9	12.3	8.1	-0.7	-0.6	-0.5
SA		8.9	7.5	72	66	71	98.7	99	98.1	9.1	8.5	8.5	8.1	4.9	9.9
TP				·	·	·	·	·	·	·	·	·	·	·	·
MEAN		6.1	6.4	74.38	73.88	75	93.43	94.55	92.5	7.3	8.5	8.88	2.15	1.39	3.41
STD DEV		2.5	0.9	10.42	9.40	9.17	5.75	8.45	5.75	2.33	3.08	1.06	3.24	2.93	4.32

O2 Fick, calculated using the Fick principle from oxygen consumption and the a-v oxygen content difference; MIG Fick, calculated using the Fick principle from the four least soluble inert gases; TD, determined by thermodilution.

Cardiac Output, Heart Rate, CVP, Mean Arterial and PA Pressures

24,900 ft
Mean PB: 283 mmHg
Breathing gas: 100% oxygen
Mask Pressure: 60 mmHg
Counterpressure: 60 mmHg
G-suit pressure: 60 mmHg

Subject	Cardiac Output O2 (l/m)	MG (l/m)	TD (l/m)	HR Start	HR Mid	HR End	Part Start	Part Mid	Part End	Ppa Start	Ppa Mid	Ppa End	CVP Start	CVP Mid	CVP End
RT		1.5		92	105	108	116	117.6	118.1	48	51.7	46.1	41.2	45.5	40.7
KK				•	•	•	•	•	•	•	•	•	55.4	52.5	•
FC		3.0	3.7	124	106	79	138.6	117.6	91.9	63.3	•	59.1	•	•	•
VW				•	•	•	•	•	•	•	•	•	•	•	•
WS			5.2	126	140	136	102	106.1	85.4	57.7	•	60.9	50.8	48.5	•
TH		4.4	6.3	143	160	170	109.4	112.5	115	55.9	•	65.1	47.1	42.7	38.4
JL		3.2	1.8	120	129	87	147.1	135.4	77.9	60.1	•	61.7	•	60.9	•
TLH		7.3	6.8	128	140	162	142.8	146.1	120.7	58.5	59.5	57.7	46.7	37.5	44.5
SA		5.2	8.8	106	127	157	126.1	137.2	120.4	55.2	59	50.1	48.8	51.8	46.6
TP		4.3		114	148	153	131.4	133.4	117.2	57.3	50.3	49.5	49.1	48.4	44.9
MEAN		4.1	5.4	119.125	131.9	131.5	126.68	125.738	105.825	57	55.125	56.275	48.44	48.475	43.02
STD DEV		1.8	2.5	15.38	19.3	35.52	16.32	14.10	17.68	4.43	4.80	6.83	4.32	7.01	3.36

O2 Fick, calculated using the Fick principle from oxygen consumption and the a-v oxygen content difference; MIG Fick, calculated using the Fick principle from the four least soluble inert gases; TD, determined by thermodilution.

Discussion

DR. STOLP: This increase in the high ventilation perfusion gas exchange units seen with PPB is likely to result in a significant discrepancy between end-tidal and arterial PCO2, so any conclusions based on end-tidal CO2 may not reflect what's really going on in the blood. This is evidence that invasive studies may be needed to truly explain what's going on in positive pressure breathing.

SQN LDR GRADWELL: As regards the oscillation in pulse pressure, would you agree that if you see a loss of variation in pulse pressure, it's indicative of a deteriorating condition and almost presyncopal?

DR. STOLP: Yes, if you see it, it's presyncopal. It's similar to the condition of being fluid depleted. We were able to reverse it somewhat by increasing the G-suit pressure. I'm sure if we would have had a full-coverage G-suit with a 2-to-1 pressure ratio, we would have either not seen it, or certainly prolonged the time before seeing it. Although we increased the ratio to 4- to-1 in our 5-bladder suit on one subject, the presyncopal symptoms reappeared after a minute or two.

COL. STORK: Your point about oxygen delivery is critical. I wondered if you measured the AVO2 content difference and what you found?

DR. STOLP: We measured the AVO2 difference, but I don't have a graph of the data.

DR. MOON: The AVO2 difference increased as you would expect with the reduction in cardiac output.

DR. STOLP: You can calculate O2 uptake using the AVO2 difference and the cardiac output. It corresponded well with the mixed expired gas data, so it followed what you'd expect.

COL. STORK: Your total O2 delivery can sometimes mask what's happening at the tissue level. I was wondering if you had any follow-on studies planned where you might look at regional models of tissue circulation.

DR. STOLP: Dr. Moon will discuss some of the future studies we have planned in his presentation.

COL. STORK: As you have noted, in our attempt not to be invasive, we frequently look at end-tidal gases and try to predict what's happening to the PAO2 during PPB. We rarely look at oxygen delivery and extraction at the tissue level.

DR. STOLP: Some of these data validate what Dr. Goodman will show later, in regard to the oscillations that we see in O2 delivery. He has a lot of data on extremely well-trained subjects, who are breathing at 80 mm Hg pressure sustained for 10-20 minutes. Those data also suggest differences between end tidal and tissue level oxygen.

COL. STORK: You had indicated that there was a decrease in cardiac output, which I assume was the result of a reduction in stroke volume. Can you give us the numbers on the effects of pressure breathing at this level, i.e., the percent decrease in venous return or stroke volume?

DR. MOON: Stroke volume was reduced more than 15%. The heart rate increased slightly and stroke volume was reduced a little more than the proportional reduction in cardiac output, so it was around 20-25%.

DR. STOLP: Mind you, we were not using a full-coverage suit and the suit-to-vest pressure ratio was 1 to 1. I'm sure the reduction in stroke volume and cardiac output would be much less if we had used the full-coverage suit with the 3-to-1 pressure ratio. However, there is a suggestion even in the newer suits that the patterns of pulse pressure during long sustained ventilation may actually be reduced towards the presyncopal level at higher breathing pressures.

DR. WEBB: Would you hypothesize on the effect of a computer-controlled chest counterpressure based on mask pressure?

DR. STOLP: Mask pressure is very critical, mask seal is very critical. Some regulators, however, are able to maintain a significant mask pressure even with a poor mask seal. We could tell when we had a significant leak because we saw central venous pressures drop. The approach currently taken with the jerkin/vest valve and breathing from the same supply line seems to be working quite well.

PROF. ERNSTING: I think you must consider the level of hyperventilation along with the degree of counterpressure. I feel it reflects on the specific protective assembly being used. In all our studies with full front counterpressure and a 1-to-1 ratio, the maximum increase we noted was a doubling in pulmonary ventilation in 20 subjects. We have since measured pulmonary ventilation in hundreds of aircrew. I think we have to be very careful about arguing from one assembly to another about the specific effects of positive pressure breathing. Positive pressure breathing in the COMBAT EDGE assembly with a 1-to-1 will not give the same data as another suit assembly.

DR. STOLP: I would agree. Along with the specific regulator as well.

PROF. ERNSTING: Yes, regulators and pressure breathing/suit ratios. However, many of us don't use regulators for ground level pressure breathing, because it is extremely difficult to design an oxygen regulator that functions exactly the same at sea level and at altitude. If you use them at sea level, you usually will get very large fluctuations in mask cavity pressures as compared with high altitude. The old trick we've always used is to pressure breathe through the wall of the chamber.

DR. STOLP: In our breath-by-breath measurements we were able to tune the regulators so that there was never more than a 4 mm Hg pressure variation at 60 mm Hg PPB. It did take us a while to get the regulators tuned for that and it's very critical.

PROF. ERNSTING: I have the data from the 1950s where we had our subjects pressure breathing at 60 to 80 mm Hg using full trunk counterpressure. Mean pulmonary ventilation increased from 8 to about 11 or 12 liters a minute. We are most concerned, as Dr. Macmillan indicated earlier, about the decrease in cerebral blood flow associated with these levels of hyperventilation.

DR. ACKLES: We haven't been measuring the CO_2 levels in our experiments, but our subjects are trained to be very conscious about hyperventilating. If anything, they usually breathe at a much lower rate.

DR. STOLP: The training in our studies involved several sessions until the subject was comfortable enough to maintain steady state ventilation's at 60 mm Hg for at least 5 minutes. Once they demonstrated the ability to maintain these levels we would select them for further study. It may well be that the patterns of breathing were different than in repeat subjects that are exposed year after year.

DR. ACKLES: We trained our subjects to slow their rate of breathing.

DR. STOLP: We let our subjects choose the pattern of breathing they could maintain for 5 minutes. We did not want to impose a pattern.

Influence of G-Suit Coverage on the Cardiovascular Response to PPB

Len S. Goodman, Ph.D.

Introduction

The deleterious cardiovascular effects of positive pressure breathing (PPB) have been examined in many laboratories, and for many years. It was established as far back as the 1940s and '50s, that increasing the intrapulmonary pressure (to ventilate the alveoli with 100% O2 during emergency exposure to low ambient barometric pressure), resulted in a disruption in venous return, leading to the chain of events that resulted in PPB-induced syncope (4, 5, 13, 14, 15). Clearly, what was intended as an emergency hypoxia countermeasure would have equally negative effects on aircrew cardiovascular function as the original hypoxia was having on blood oxygen status. Ideally, successful "get-me-down" protection entails providing a constant flow of oxygenated blood to the central nervous system, in the face of loss of cockpit integrity and exposure to extreme hypobaria. With the introduction of the F-22 and Eurofighter 2000, the reality of routine training or wartime flight above 60,000 ft will demand that aircrew are protected not only for "get-me-down" protection as in the past, but possibly even "mission-completion" scenarios after decompression. This will necessitate the tolerance to long-duration/high levels of PPB.

Thus, the intent of PPB is to deliver high pressure 100% O2 to the respiratory tract, preventing profound hypoxia at high altitudes (> 40,000 feet), while simultaneously minimizing the negative cardiovascular effects, which themselves would negate the countermeasure's effectiveness. Thus, the anti-G suit design is critical to the success of PPB for emergency high-altitude protection, by preventing large reduction of effective central blood volume due to pooling. I will first summarize, historically, our research efforts at DCIEM studying expanded-coverage G-suits, and conclude with discussions relating to recent efforts to modify the G-suit inflation schedule when using modern full-coverage garments.

Background

The introduction of the anti-G suit, originally conceived for +Gz protection, was combined with upper body counterpressure garments, to form the emergency high-altitude ensemble for PPB. The British developed "Jerkins" and "Waistcoats," that pressurized the whole trunk, including the abdominal and pelvic regions, and combined variations of these with an anti-G suit. The Canadians, Swedes and Americans retained a minimal-coverage "Vest" concept (originally introduced by the Scandinavians) for the upper body garment, while relying exclusively on the anti-G suit for the lower body and splanchnic counterpressurization.

The CSU-type anti-G suits, still used in most North American Air Forces, have not changed appreciably since World War II. This cut-away design, containing bladders that pressurize about 40% of the lower body, has been the life-support workhorse for many years. Work done at DCIEM in the 1970s using a G-suit of this vintage was used in the original "Phoenix" experiment series, and human subjects were successfully explosively decompressed in the hypobaric chamber to 80,000 feet for one minute (20). However, to provide the necessary counterpressure to prevent loss of central blood volume, the G-suit had to be pressurized to multiples of the breathing pressure.

Ackles et al. (1) demonstrated that stroke volume, though reduced during PPB as a function of G-suit/thoracic coverage used, was reduced the least when the G-suit was pressurized to 4 times breathing pressure (Figure 1). Thus with a mask cavity pressure of 70 mmHg (for emergency protection at 60,000 feet), the G-suit would be pressurized to 280 mmHg. Work done by Balldin (2) about the same time, concluded the same, the G-suit
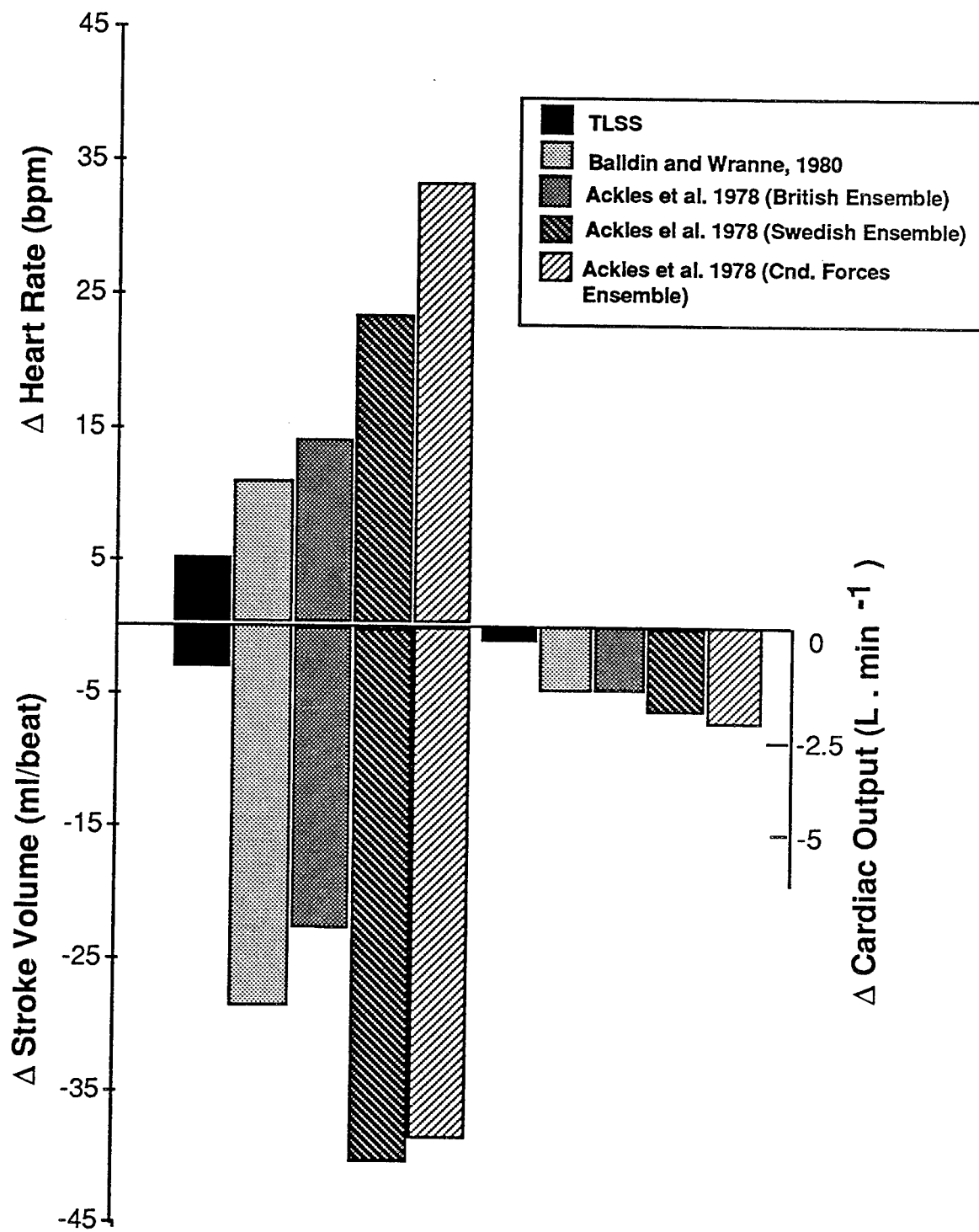
Figure 1. Comparison of 4 minutes 70 mmHg PPB for TLSS vs Others.

Figure 2. Tactical Life-Support System (TLSS) ensemble.

suit should be inflated to 3.2 times the breathing pressure. Another paper by Balldin and associates was the first to use invasive measurements, and was a landmark study in our understanding of the central hemodynamics during the stress of PPB (3). Since that time, a recent and comprehensive study has been performed using invasive techniques to measure detailed cardiovascular and pulmonary effects of PPB (26). Both of these studies confirm the fact that PPB causes profound pooling of blood in the dependent regions of the body, changing central hemodynamics. The group from Duke University (26), has also added to this finding by describing the significant ventilation/perfusion mismatching in the lung during PPB, causing impairments in oxygen delivery.

During the 1970s, it was apparent that due to the sudden increase in aircrew losses, aerospace physiologists would have to devote increased attention to the problem of G-induced loss of consciousness. About this time, the application of the anti-G suit for +Gz protection became dominant, while the operational role of high-altitude flight diminished somewhat with the introduction of satellite surveillance technology and continued use of reconnaissance U2 operations using full-pressure suits. Thus, the role of the G-suit (originally developed in the 1930s as a countermeasure against +Gz) had largely been adopted by the acceleration physiology community as a method of supplementing head-level blood pressure by directly increasing systemic vascular resistance. However, the defence of venous return during +Gz stress has been viewed as a minor role of the G-suit. This has stemmed largely from earlier work demonstrating that heart-level blood pressure was reasonably well maintained at +Gz (8, 24, 25).

Conversely, in +1Gz PPB applications, the G-suit must function entirely as a venous return-assist device. The elevation in blood pressure with PPB is a nuisance and unavoidable side effect when intrapulmonary pressure is increased, and does not positively affect the end aim--hypoxia reversal. However, its reduction during PPB is a harbinger of uncompensated cardiovascular failure and immanent syncope (10, 11, 12, 14). Paradoxically, it may turn out that the added systemic vascular resistance and blood pressure arising from PPB might act in preventing decompression sickness by imposing a generalized whole-body hyperbaria, thereby reducing venous gas emboli formation while at altitude (Personal communication, Fraser, W.D., 1995).

In any case, the central role of preserving venous return (or more precisely, enhancing venous outflow from dependent regions where pooling has occurred), in order to maintain cardiac filling pressure, must be addressed. At DCIEM, the next research effort was to expand the coverage of the G-suit. This was embodied in the Tactical Life-Support System (TLSS) ensemble (Figure 2), that, in addition to high-altitude protection (offering PPB), would protect against +Gz (using PBG), NBC, cold/thermal stress and immersion. TLSS incorporated integrated upper- and lower-body partial-pressure garments, and was intended to act as a one-piece, multi-function/countermeasure garment. From a PPB standpoint, the bladders in the TLSS G-suit were increased to pressurize an additional 40-45% of the legs. The abdominal bladder was also increased in size in some versions. Expansion of the bladders resulted in a circumferential coverage design about the legs, effectively applying a more uniform pressure distribution.

The advantages of TLSS over the standard CSU-variant G-suits during PPB were borne out in several research efforts. The decrement in estimated stroke volume (measured by impedance cardiography) was significantly lower using TLSS over 10 minutes of PPB. Furthermore, very high PPB levels (88 mmHg) were tolerated by subjects for an entire 10 minutes (Figure 3) when wearing the TLSS (vs. standard G-suit) (16). Conversely, the standard CSU-13 G-suit resulted in a markedly reduced stroke volume and blood pressure in most subjects at this PPB level, and few subjects could tolerate the entire 10-minute exposure.

With regard to the performance of the TLSS garment, stroke volume results (using impedance cardiography) were interesting. They suggested that there was a transient increase in stroke volume within the first few seconds of PPB, followed by a maintenance or slight decrease. Stroke volume is remarkably well maintained using TLSS vs. a standard G-suit. However, a sudden rise in SV is unexpected, especially since R. atrial pressure is immediately increased with the onset of PPB. It is possible that this transient effect is due to one of the following: (1) a squeezing of blood out of the low-resistance pulmonary circulation secondary to the rise in intrathoracic pressure, increasing left atrial pressure, left ventricular preload and stroke volume according the Starling Law. In addition, the expanded-coverage G-suit increases right atrial pressure, further increasing preload in advance of any

significant pooling; (2) a sudden rise in right atrial pressure with the onset in PPB transiently and markedly reduces right ventricular filling, and immediately removes a large amount of blood in the pulmonary circulation. This causes an immediate carotid baroreceptor reflex that increases peripheral resistance, blood pressure, and left ventricular contractility, resulting in an increased stroke volume until an equilibrium is reached in peripheral and central venous pressures. These two proposed physiological events are obviously entirely divergent. Without the use of more sensitive measure of left ventricular function and baroreceptor monitoring, however, we cannot be sure what the true transient events are during PPB.
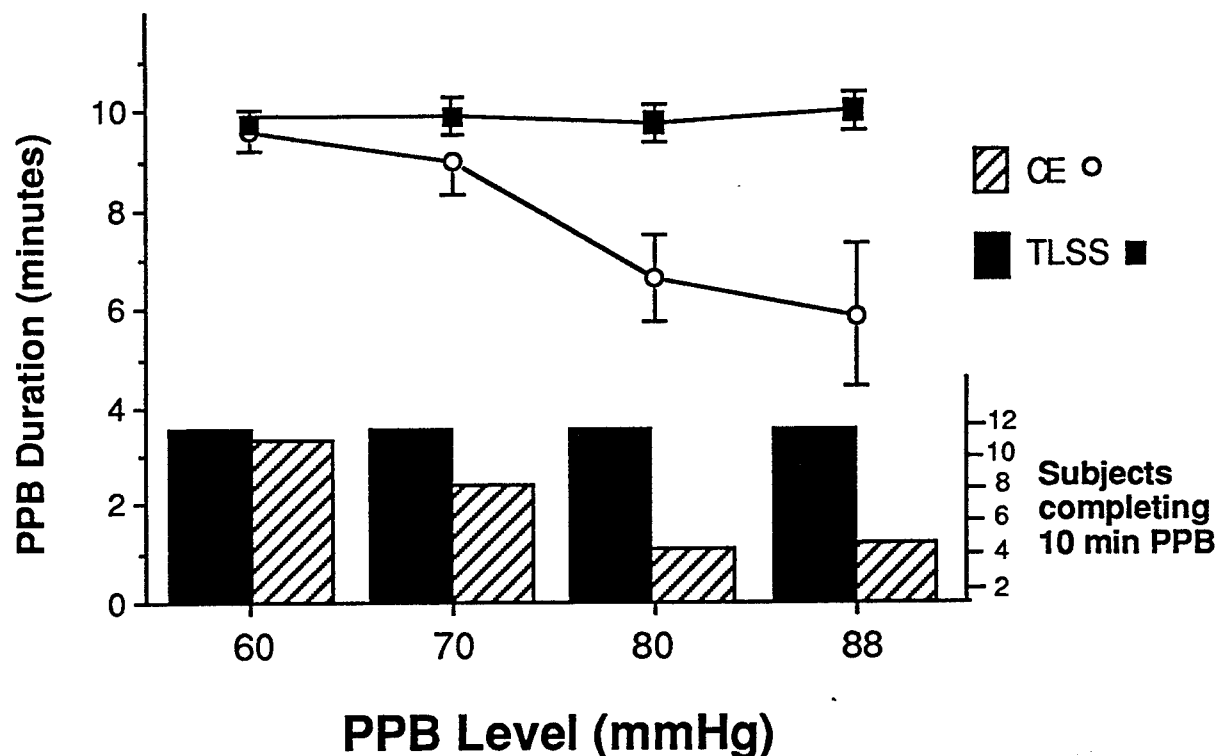

Figure 3. Comparison of PPB Tolerance, TLSS vs standard G-Suit.

Certainly, there is probably a competition between the intra-thoracic (aortic) and extra-thoracic (carotid) baroreceptors during PPB, which continues throughout the exposure. Regardless, it was very evident that increasing the coverage of the G-suit had dramatic events on PPB tolerance and physiology, with the defence of central and intracardiac blood volume.

The next effort was to examine the hypothesis that indeed stroke volume was maintained during PPB by virtue of maintained left ventricular filling, which was in turn a function of G-suit coverage. In the past, we have used the technique of impedance cardiography for the measurement of stroke volume/cardiac output (21, 22). It offers many advantages: safety, non-invasive, ease of use under partial-pressure garments, and provides continuous beat-to-beat data. However, it does not supply information concerning the dynamics of left ventricular function, particularly filling rate, end-diastolic volume, and ejection characteristics. These events, particularly the diastolic filling aspect, are of particular importance, since changes in left ventricular filling (according to the Starling law of the Heart) will, in healthy individuals, reflect any changes in venous return to the heart.

Echocardiography has been attempted in this and other laboratories, but it necessitates modification to the upper pressure garment, and a skilled operator to manipulate the ultrasound probe. We instead pursued the nuclear cardiology field, and began investigating the use of a miniaturized left ventricular function monitoring system, Cardioscint™ (Figure 4). The system uses a small single cesium-iodide photodiode probe that is placed over the heart, directly on the chest, after the subject's red blood cells are labeled with radionuclideTechnetium 99m. This is

done with a small injection into a forearm vein. The left ventricle's position is determined by a previous gamma camera scan, or by the Cardioscint computer's algorithm, which displays a graphic representation showing counts as the probe is moved over the chest. The system has been validated against recognized gold standards with good results (6, 7).

$$\text{LVEF (\%)} = \frac{\text{EDV counts - ESV counts}}{\text{EDV counts - Background counts}} \times 100$$
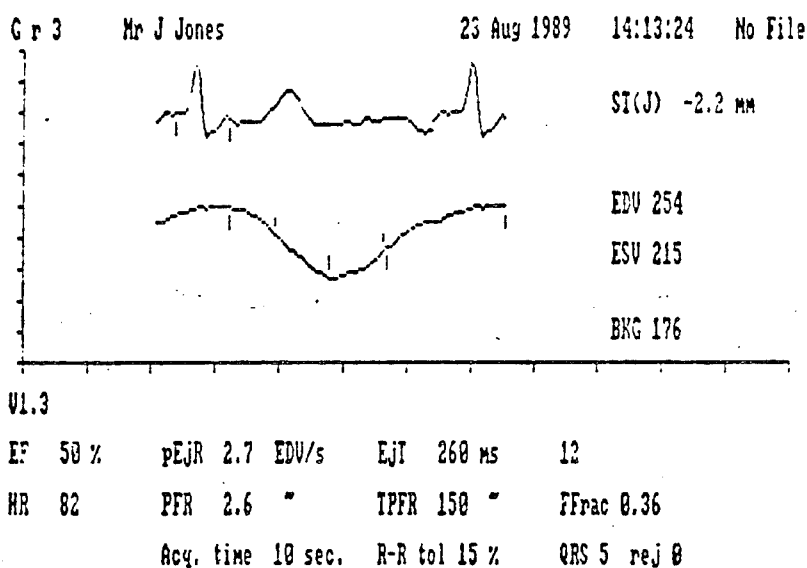
Figure 4. Cardioscint™ with time-activity curves.

The Cardioscint's probe has a conical field-of-view, and consequently position changes of the heart (which do take place during PPB according to some unpublished preliminary echocardiography trials conducted at DCIEM) do not jeopardize the accuracy of the measurements. The background counts are continuously subtracted, and the physical decay of the isotope is also corrected continuously. A continuous series of time-activity curves (Figure 4), are generated. The computer counts the number of cardiac cycles occurring within continuous 10 sec sliding windows of time, and then ensemble averages these individual time-activity curves to generate one representative smoothed curve for each 10 sec period. Indices such as relative end-diastolic volume, end-systolic volume, peak filling rate, ejection rate, and left ventricular ejection fraction are calculated in real-time from these time-activity curves, are briefly displayed on the screen and simultaneously stored in memory. A real-time trend plot may also be viewed on the screen or printed after the study. The end-diastolic volume is calculated from the absolute maxima of the count/time curve; end-systolic volume is calculated from the absolute minima of the count/time curve, and rates of ejection and filling are calculated by an algorithm that determines the maximal rate of change of the upslope (diastole; filling) and downslope (systole; ejection) phases of the curve.

The system continuously measures changes in counts within the left ventricle, and does not construct a picture of the heart, as standard gamma camera systems do. Consequently, the absolute volumes are not available, and indices like end-diastolic volume are expressed as absolute counts, and are relative measures. Rate and ratio measures however, are identical to those obtained from a dedicated imaging system. For example, left ventricular ejection fraction is calculated by the standard equation: LVEF = (end-diastolic volume counts--end-systolic counts)/(end-diastolic counts--background counts) x 100. Therefore, this ratio is equal to that calculated from imaging studies. The advantage of the non-imaging Cardioscint system is in its ability to measure rapid changes in left ventricular function; standard image systems required at least 2 minutes of acquisition to form a static or dynamic moving image of the left ventricle. This is suitable for clinical studies, but during PPB, many transient events would be missed when averaged over this long period.

The detector itself is small and lightweight, easily fitting on the chest, and under the flight suit and counterpressure garments. We use a foam donut surrounding the probe to minimize pressure points. Slippage on the chest is eliminated by the use of 3M Stomoseal double-sided adhesive applied directly on the probe's flanges.

We essentially repeated the same previous experiment comparing the TLSS vs. standard G-suit during PPB, but with the use of Cardioscint to measure left ventricular filling differences (17). These experiments were done in a hospital setting, using a pressure panel for the G-suit and breathing pressure. Figure 5 shows that heart rate was elevated progressively with the standard G-suit at 70 mmHg PPB, with very little change when using TLSS. These differences in heart rate response had been observed in previous studies studying the same garments. The new information concerning left ventricular filling, however, reflected what had been observed using impedance cardiography to measure stroke volume. Figure 6 is a plot of the changes in relative end-diastolic volume (EDVr) against time at two PPB levels, 30 and 70 mmHg. It is apparent that the initial fall in EDVr is rapid, followed by a plateau. The fall using TLSS is half that when using the standard G-suit, indicating a superior defence of left ventricular filling, and we have concluded that this is indicative of the efficacy of the G-suit to minimize dependent blood pooling, thereby better preserving central blood volume.

These data also suggest that myocardial contractility and coronary blood flow is not compromised during PPB. This is evident by examining peak ejection rate, which did not change during PPB at any PPB level. The heart does become smaller during PPB, especially when using a standard-coverage G-suits. This was confirmed by imaging studies with the gamma camera system prior to the use of the Cardioscint. However, this does not seem to negatively influence myocardial energetics during PPB. The ECG S-T segment depression is also continuously analyzed by the Cardioscint, and there were no changes in any subjects during PPB.

Current Status

With the introduction of various hybrid full-coverage anti-G suits, the level of cardiovascular protection for both PBG and PPB has improved markedly. We have currently been evaluating versions of the NAVY Eagle,

USAF Advanced Tactical Anti-G Suit (ATAGS), and Canada's STING (Sustained Tolerance to Increased G) anti-G suit in both +Gz and PPB-altitude environments. The additional bladder volume and coverage offered in these garments improves counterpressure "footprint", and comfort by further increasing the bladder coverage over the legs and calf, but also the extent and effectiveness of circumferential coverage. The abdominal bladders have remained roughly the same size, as some abdominal discomfort has been reported with increased coverage over this area. The ATAGS suit also enables pressurization of the feet, which though not offering a large volume of pooled blood, will become increasingly engorged, and cause significant pain and discomfort in some individuals when the garment is inflated to high pressures without foot counterpressure.
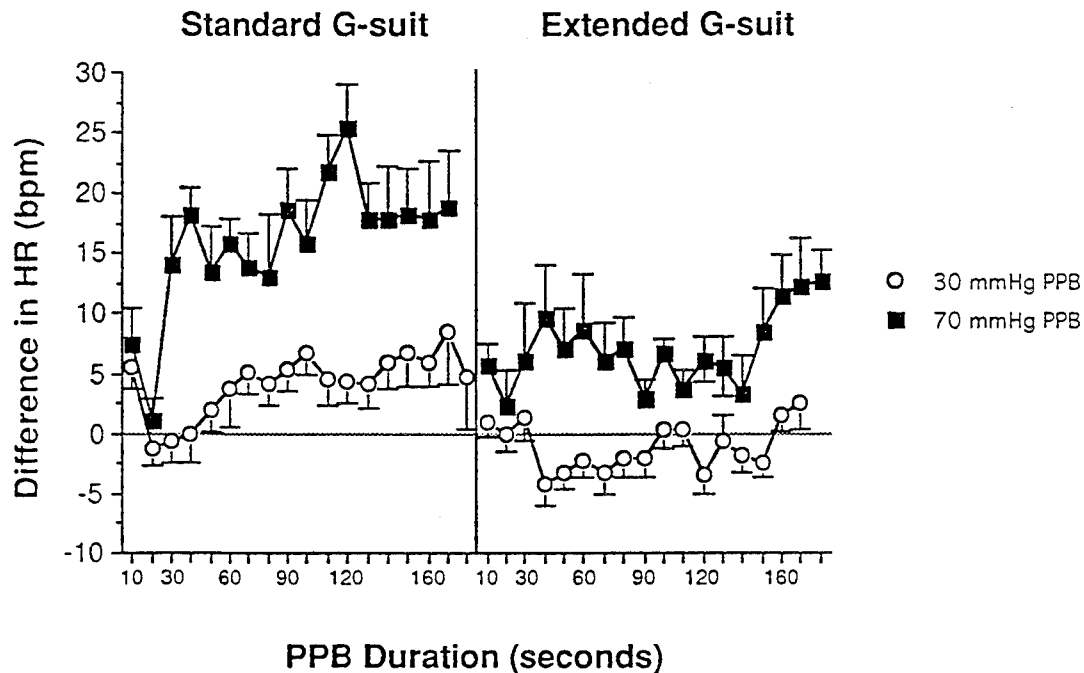

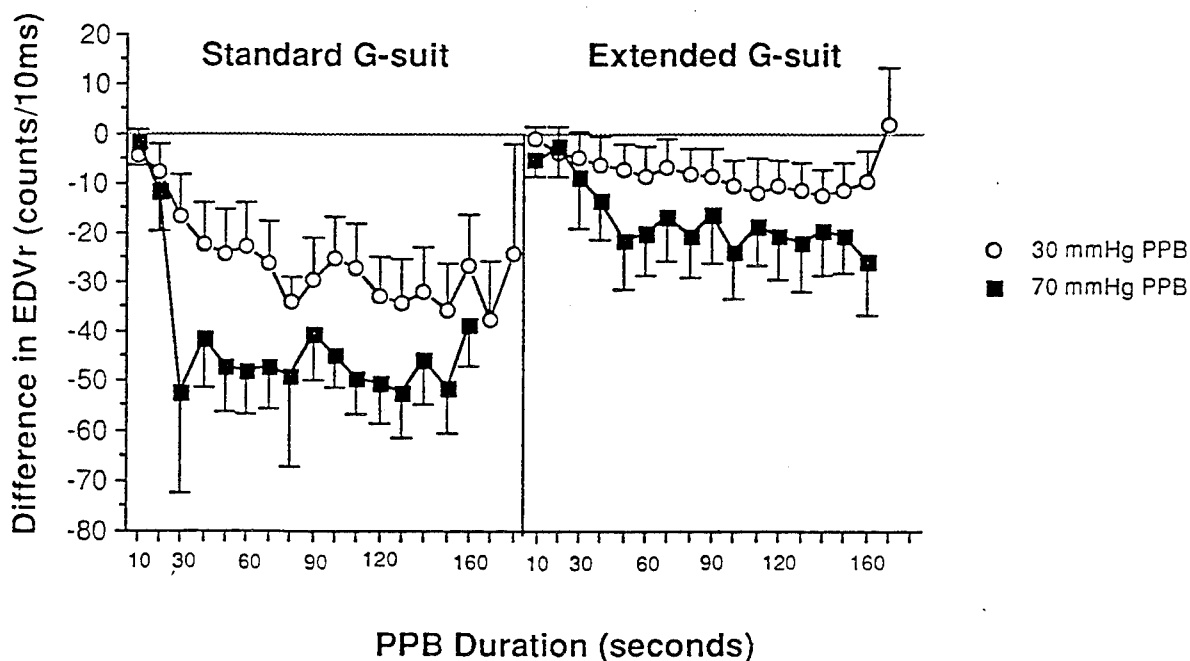Figure 5. Heart Rate Comparison, Standard G-Suit vs. Extended G-Suit.

Figure 6. Changes in EDVr at two PPB levels, Standard G-Suit vs. Extended G-Suit.

In recent experiments testing the efficacy of these full-coverage G-suits during explosive decompression and ground-level experiments, it became evident that when many subjects performed PPB at high levels using the standard 4:1 G-suit to breathing pressure ratio, heart rate immediately slowed to rates below the previous control condition. Often, the lower heart rate persisted throughout the entire PPB period. Figure 7a illustrates blood pressure response (Finapres) before and within the first few seconds of PPB when wearing the Combat Edge G-suit inflated to 280 mmHg (4:1 ratio) at 70 mmHg PPB. Although heart rate is slowed transiently (i.e., through a baroreflex), it quickly increases to compensate for reduced stroke volume. In contrast, Figure 7b shows that with the ATAGS garment at the same PPB level/G-suit pressure ratio, heart rate slows persistently after PPB is applied.
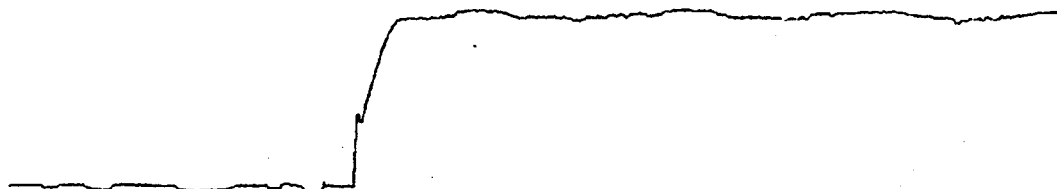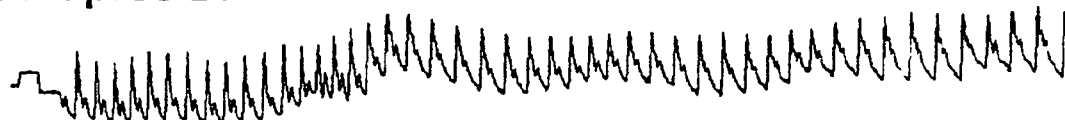
Finapres BP
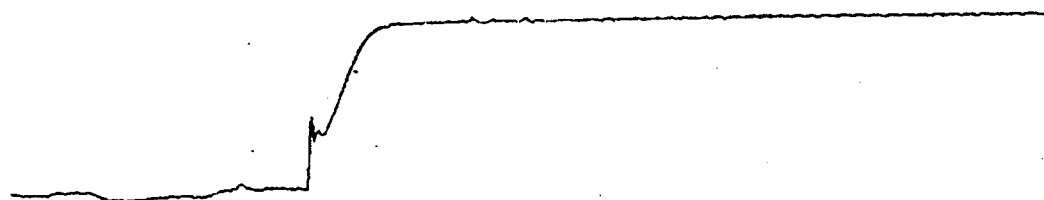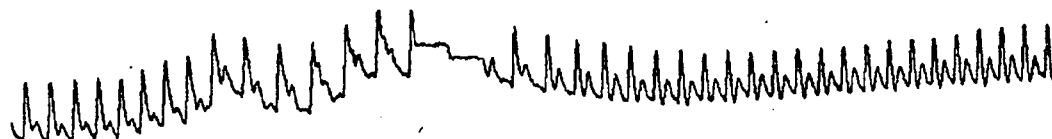
mask cavity pressure

Figure 7a. Blood pressure response—Combat Edge G-Suit.

Finapres BP

mask cavity pressure

Figure 7b. Blood pressure response—ATAGS garment.

This observation lead to experiments attempting to test the hypothesis that full-coverage G-suits employing the same 4:1 inflation ratio result in an overpressure condition, manifesting in reduced heart rate response and possibly adverse effects such as baroreflex-induced peripheral vasodilatation. Furthermore, recent evidence that right atrial stretch receptors are also involved in cardiodepressor and vasomotor reflexes required this question to be addressed. Finally, the 4:1 ratio in the ATAGS garment is also uncomfortable for some, especially in the unpressurized feet and abdominal region. Alternatively, we speculated that the pressure ratio could be reduced significantly below 4:1 when using ATAGS, which would afford similar cardiovascular protection, but with more comfort and physiologically appropriate cardiovascular response in a hypobaric environment.

This experiment also provided the opportunity to again use the Cardioscint left ventricular function monitor system to test the hypothesis that surplus venous return to the left ventricle was occurring, resulting in excessive blood pressure rise and reduced heart rate (18, 19). In addition to Cardioscint, impedance cardiography was simultaneously employed. Seven subjects received the radionuclide RBC label and an initial cardiac scan, then were transported back to DCIEM for the experimental session. Each subject performed PPB at 60 mmHg, under six individual conditions, with rests in between: Combat Edge (i.e., standard CSU-13/P G-suit with standard pressure vest) at 4:1 ratio, TLSS at 4:1 ratio, ATAGS at 4:1, 3:1, 2:1 and 1:1. Each PPB exposure was 2 min. duration. The Cardioscint was mounted to the chest with the special harness after impedance cardiography leads were attached, and then the upper vest pressure garment was donned. The subject was then inserted into the altitude chamber. PPB was produced using the "through the wall" technique, exploiting the pressure differential between the ground and the reduced altitude chamber pressure when ascended. Mask/jerkin pressurization occurs once a valve is opened across the chamber wall, allowing air to enter the inlet hose. G-suit pressure was controlled separately from a compressed air tank/regulator.

The subjects rested/recovered for 10-20 minutes between runs, and baseline conditions were established using a combination of real-time heart rate and left ventricular ejection fraction criteria from the Cardioscint. The entire 6 PPB exposures were completed in about 3-4 hours per subject. To avoid confounding the four ATAGS conditions by introducing possible garment tightness variations, it was decided to impose a modified randomization of the garment conditions. This was done by randomizing the order of Combat Edge/TLSS/ATAGS conditions; within the ATAGS conditions, the order of the four experimental pressure ratios were themselves randomized.

Heart rate was reduced by 9 and 7 bpm during the ATAGS 4:1 and 3:1 conditions, respectively. There was no change from the control period at the ATAGS 2:1 condition, but it was elevated significantly by 9 bpm at the 1:1 ratio. The Combat Edge condition resulted in a 18 bpm rise in heart rate, and PPB using TLSS caused a small 1 bpm decrease in heart rate from control conditions.

Figure 8 plots base thoracic impedance (½) against PPB time for the six G-suit coverage/pressure conditions. The ½ measure roughly reflects blood content in the thorax, and will increase when blood volume is reduced, and decrease when blood volume is increased (as red blood cells are better conductors than other tissues). It can be observed that the ATAGS 4:1 condition causes the lowest ½ reading throughout PPB, suggesting that blood volume in the thorax is the highest. In fact all of the ATAGS 4:1, 3:1, 2:1, and TLSS conditions reflect at least equal or greater thoracic blood volume during PPB vs. baseline, suggesting that venous return is at the very least, defended adequately. Only the Combat Edge (CE) and ATAGS 1:1 conditions result in elevated ½ (i.e., reduced thoracic blood volume) during PPB.

Calculated stroke volume from the impedance cardiography data set are presented in Figure 9. Again, stroke volume is well preserved, even above baseline conditions throughout PPB in all ATAGS conditions (except 1:1), and with TLSS at 4:1. The ATAGS G-suit inflated to only 60 mmHg during 60 mmHg PPB (i.e., a ratio of 1:1) is equally ineffective as the Combat Edge G-suit in preventing reduction of stroke volume throughout PPB.
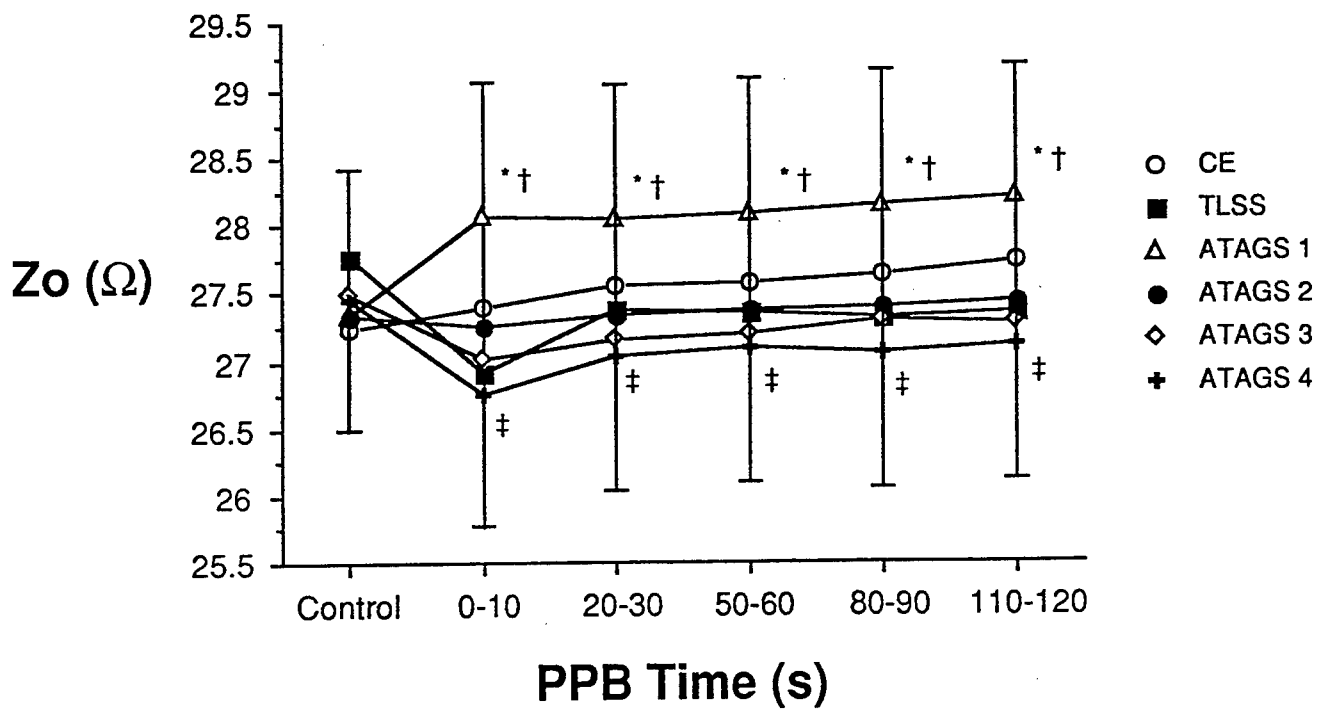
Figure 8. Base thoracic impedance (½) against PPB time for the six G-suit coverage/pressure conditions.
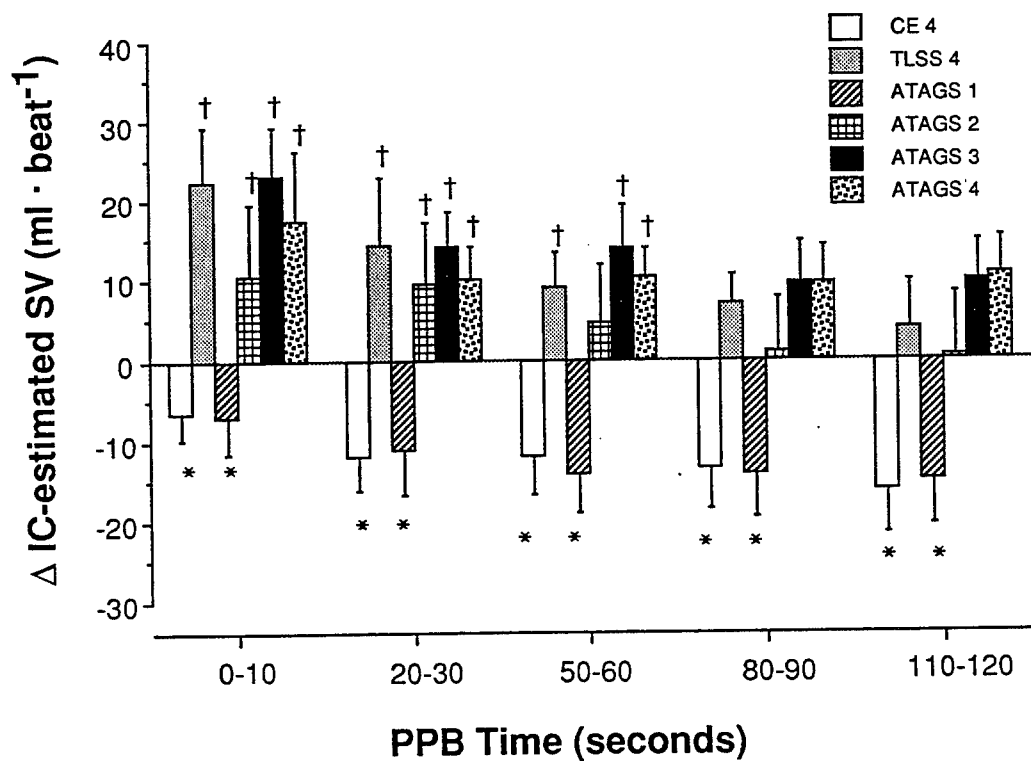

Figure 9. Calculated stroke volume from the impedance cardiography data set for the six G-suits.

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The Cardioscint left ventricular function data is consistent with the heart rate and stroke volume data. Figure 10 illustrates the change in relative EDV, averaged over the entire 2 minutes of PPB for all subjects, and shows that the greatest (and only significant) fall in left ventricular preload was with the Combat Edge (at 4:1) and ATAGS at 1:1 ratio. The other G-suits sustained small, but non-significant reductions. A more revealing observation of left ventricular peak filling rate (pfr; during the rapid filling phase in diastole) is presented in Figure 11. It can be observed that the greatest coverage garments allow a transiently raised pfr in the immediate first 10 sec. of PPB. Although reduced somewhat throughout PPB, pfr is well maintained by 120 sec, whereas with the ATAGS inflated at 1:1, and Combat Edge inflated at 4:1, pfr is significantly depressed by the 120 sec mark.
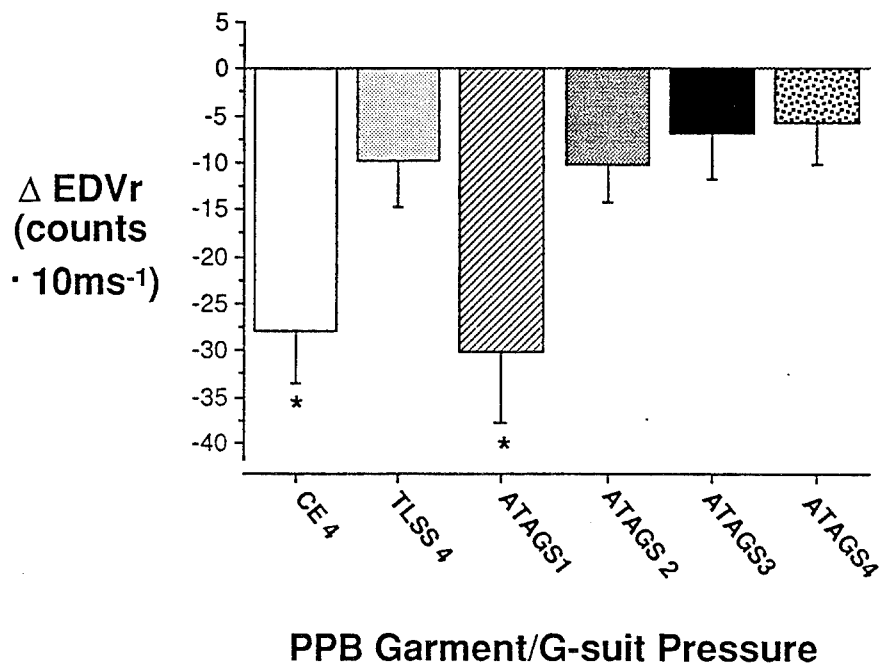

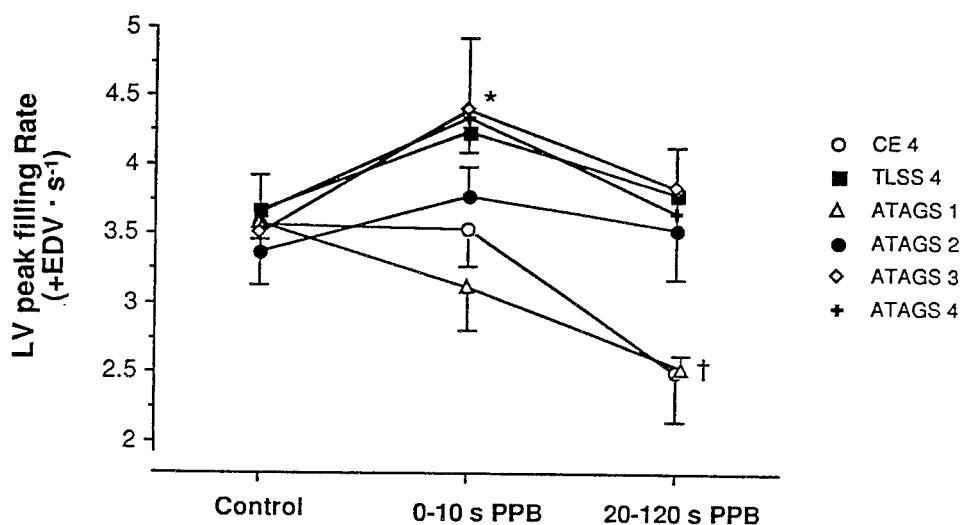**Figure 10. Change in EDVr, for the six G-suits.**

Figure 11. Left ventricular pfr for the six G-suits.

Interestingly however, systemic vascular resistance (using calculated cardiac output from impedance cardiography and mean arterial blood pressure from the Finapres) is raised relative equally with all garment/pressure conditions. The ATAGS 4:1 condition presents the "flattest" response however, increasing initially, but maintaining, or slightly reducing throughout PPB (Figure 12). One might assume from this that though the heart is indeed well filled, and that venous return is overly adequate using the full-coverage/high-pressure suit conditions, vascular resistance is being attenuated. We propose that some still not completely understood central cardiovascular reflex is interacting, causing some reduction in arteriolar tone secondary to hyper-increased venous return and right atrial stretch. The carotid baroreceptors might also be augmenting the response. Indeed, if we plot the change in mean arterial pressure vs. changes in heart rate during PPB with all garment conditions, Figure 13 is constructed. The relationship between blood pressure rise and reduction in heart rate follows for the highest coverage/pressure G-suit conditions, as it does conversely with reduced blood pressure and elevated heart rate for the lowest coverage/pressure garments.
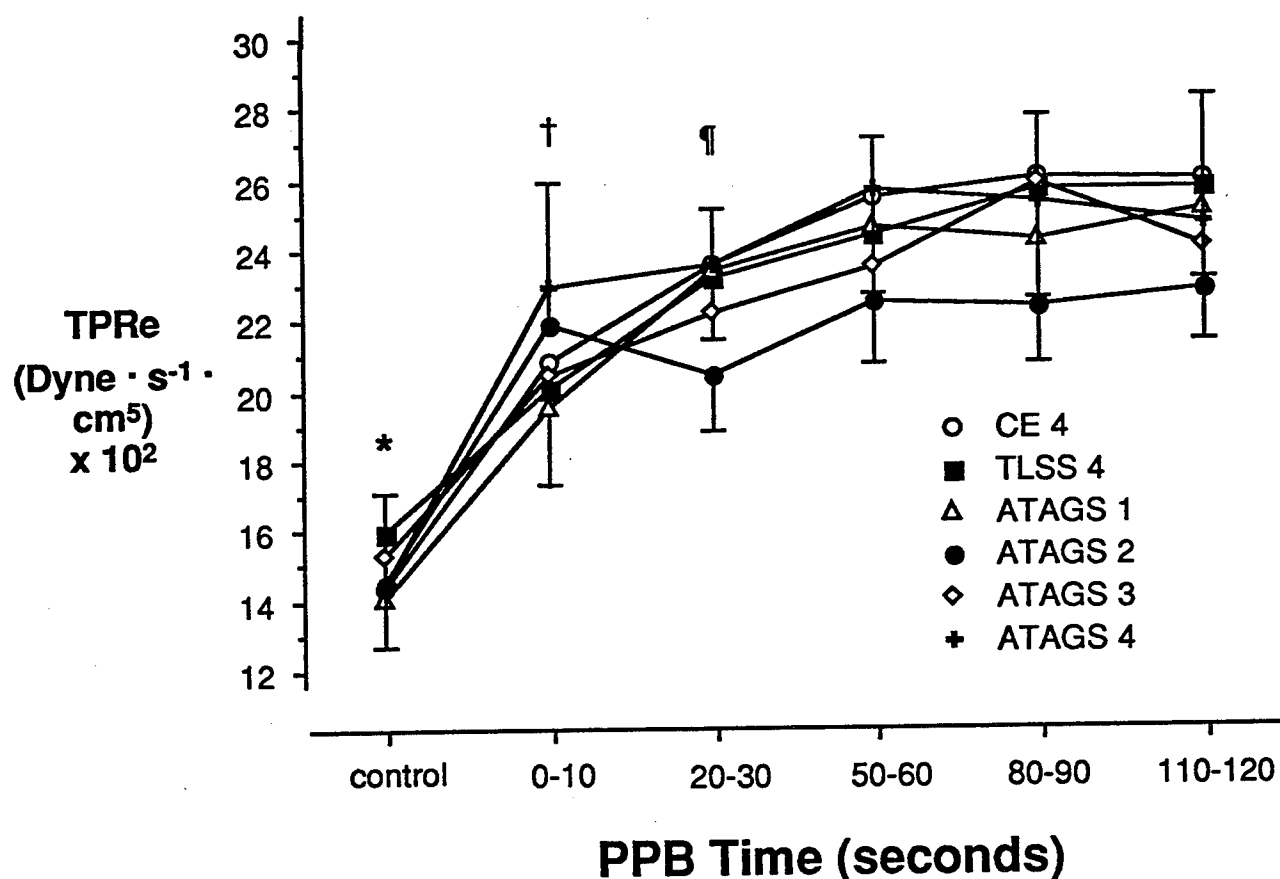
Figure 12. Systemic vascular resistance over time.

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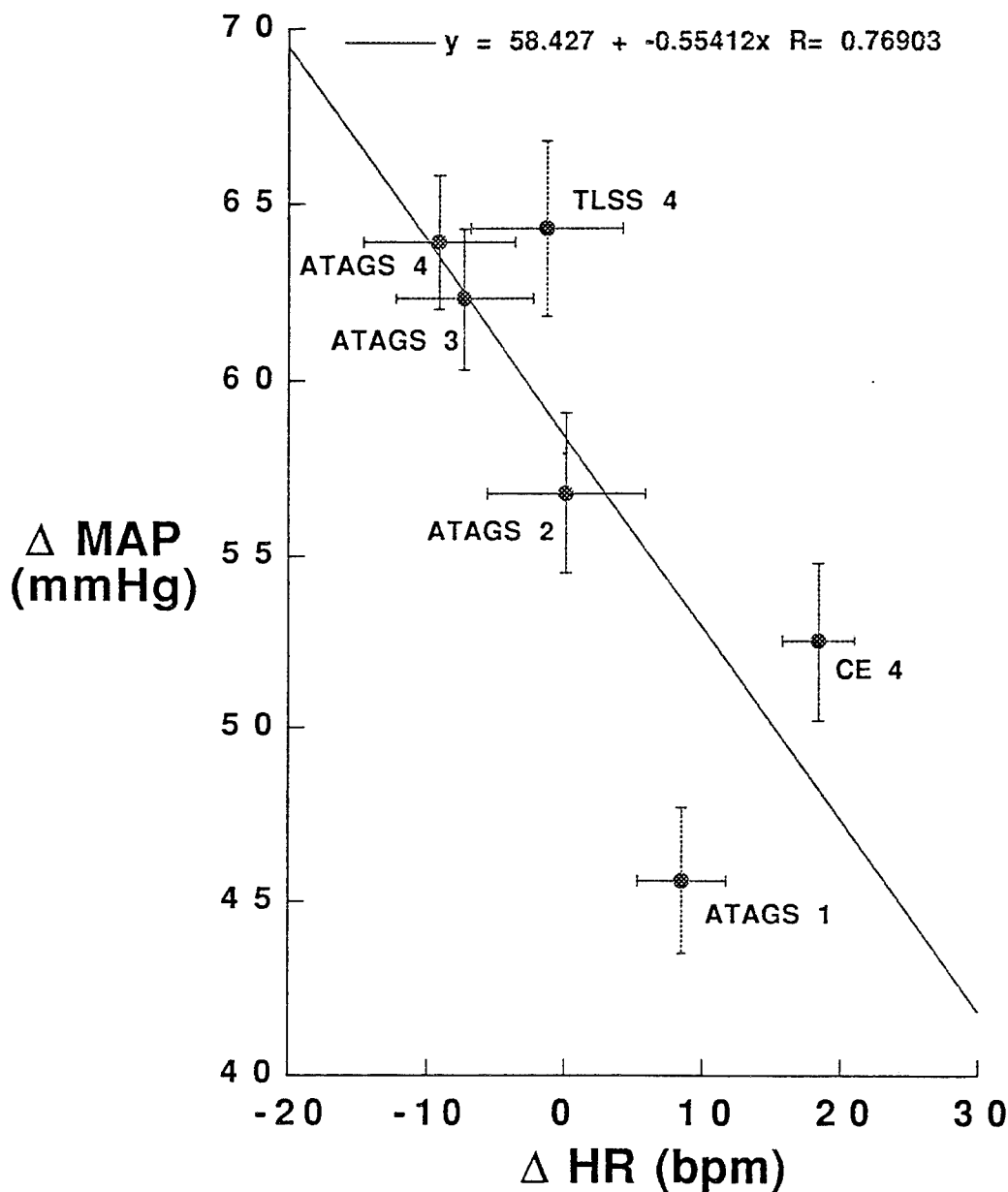
Figure 13. Mean arterial pressure vs change in heart rate with different suit ratios.

Conclusions/Recommendations

Results from recent RAF (9) and Swedish reports (23) also confirm our findings. In the RAF study, blood pressure and heart rate were measured during various gradations of inflation ratios (2:1, 2.5:1 and 3:1) during 60 mmHg PPB using the RAF Full-Coverage Anti-G Trousers (targeted for use in the Eurofighter 2000). The authors showed that the 3:1 pressure ratio condition caused a slowing of heart rate, and that a 2:1 ratio provides equal cardiovascular protection.

Thus, it appears that there is a stepwise relationship between cardiovascular protection from the effects of PPB, and the coverage and pressure of the anti-G suits used to defend venous return and blood pressure control. Using a combination of nuclear cardiology techniques and impedance cardiography, we have demonstrated that excessive pressure in full-coverage G-suits might be occurring when using the traditional 4:1 pressure schedule, originally developed and proposed for use with the older CSU-13 type G-suits. The new full-coverage G-suits afford vastly superior bladder coverage over the limbs, optimized pressure transmission to the tissues, and consequently do not require inflation pressure of this magnitude. It appears from our data, that a pressure ratio of 2:1 or 3:1 would suffice for most individuals. This level of pressurization still affords adequate left ventricular filling (and consequently, maintained stroke volume), while allowing a more appropriate heart rate response. Furthermore, there is some concern that over-pressurization of the G-suit might actually invoke undesirable skeletal muscle microvascular vasodilatation effects due to stretch of right atrial and carotid baroreceptors. With the possible use of extended duration PPB in the future (for mission-completion and/or DCS prevention purposes), the comfort factor during PPB will also now become a relevant issue. Lower inflation pressures without loss of physiological protection will address this concern.

There are some provisional considerations however. We still cannot confirm whether these conclusions would apply in an actual hypobaric condition (i.e., during explosive decompression). Therefore, further trials using these pressure ratios in actual hypoxic conditions are required before advocating the use of these reduced inflation pressures in future high-altitude tactical fighter operations. However, it seems reasonable that during the physiologic stress of a decompression, O2 delivery is augmented by encouraging an appropriate (but not excessive) increase in circulatory activity (i.e., sympathetic stimulation), vs. encouraging a slowing or hypokinesis of the circulation (by stimulating parasympathetic nervous system responses). This question will be addressed by continued research efforts in the DCIEM altitude chamber.

We have since upgraded the Cardioscint's temporal resolution capabilities with custom software. This now enables measurement of left ventricular performance and volumetric parameters in nearly beat-to-beat time periods. This will be used in subsequent studies examining transient cardiac events within the first few seconds of PPB and +Gz. Current validation of its accuracy and reliability against other gold-standard techniques is occurring presently.

Continued work needs to be addressed in explaining the function and the role of the microcirculation during the stress of PPB. This will be addressed by borrowing techniques from the cardiovascular regulatory physiology field, using techniques such as spontaneous baroreflex response with neck cuff positive and negative pressure devices, as well as the use of impedance plethysmography to determine the skeletal muscle circulatory kinetics during PPB.

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Discussion

DR. GOODMAN: There are two questions that arise with the use of a full-coverage suit: whether the PPB, necessary after a decompression at high altitude, will change baroreceptor function enough to impact the aircrew ability to withstand high G, i.e., slowed heart rate and vasodilation, and whether the G suit pressure can be reduced sufficiently to provide a far longer exposure to the higher altitudes? Indeed, will the pilot ever be exposed to high G after loss of cabin pressure?

MAJ. NEUBECK: If you leave the sanctuary of high altitude after losing cabin pressure, you will be descending to a lower altitude where there are more aircraft and other threats. There's also a higher probability of being detected, which increases the chances that you will have to react to a threat. So, yes, Gs are probably going to have to be pulled if you're still in the threat environment.

DR. GOODMAN: Then that's a possible scenario. Perhaps the pilot could be compromised if he has to pull G, after a period of pressure breathing?

MAJ. NEUBECK: The chances are increased that you're going to have to pull Gs to react.

LT. COL. DEMITRY: The logic there is compelling. The scenario gets even more complex from a physiological standpoint because, as he's pulling Gs, there's probably going to be a catecholamine and an adrenergic response to consider as part of the scenario.

DR. GOODMAN: Those responses might make it a nonissue.

LT. COL. DEMITRY: These kinds of scenarios are key from an operational standpoint. What happens in the laboratory may be of interest academically, but from a pragmatic standpoint one needs to consider these operational issues.

DR. GOODMAN: Although these points may be very esoteric, with an increased suit coverage you might be able to reduce the pressure, but if it impacts upon subsequent G performance, other approaches may be required in the development of the suit assembly.

LT. COL. DEMITRY: There is a need to get some of these numbers, because I can tell you when you really feel threatened there is a pervasive change in one's affect.

MAJ. NEUBECK: Dr. Goodman, I'm not sure I understand your second question.

DR. GOODMAN: I was wondering whether long-duration positive pressure breathing is going to be a realistic scenario. If it isn't required, then we only need to consider 30-60 seconds of get-me-down protection which we have always felt we needed. If there's a need to positive pressure breathe for a prolonged period, then this reduced G-suit ratio might have greater impact.

MAJ. NEUBECK: Are you talking about after loss of cabin pressure?

DR. GOODMAN: Yes. Do you need to continue flying at high altitude after decompression?

MAJ. NEUBECK: I think the answer to that is yes, but as I have noted, the tactical environment is very dynamic and you never really know. The likelihood of pulling high G for a long period after loss of pressure at low altitude is high.

DR. GOODMAN: That's significant because you don't want to be turning on your parasympathetic system and slowing your heart rate down and opening up the microcirculation in your legs.

MAJ. NEUBECK: When I say a long period of time, I mean two to three minutes or so.

PROF. ERNSTING: Weren't you meaning positive pressure breathing from an altitude protection point of view? The question was after a rapid decompression do you intend staying above 40,000 feet pressure breathing?

DR. GOODMAN: That's correct. If you are up at 60,000 feet and you have a decompression, and if you wanted to continue flying at that altitude for some mission requirement using positive pressure breathing to keep you oxygenated, at the same time maintain cardiovascular function so that you could continue whatever task you needed to do for greater than three, four, five minutes. Then fine tuning and optimizing the G suit pressure to the optimum ratio and coverage might have more impact for the longer exposures. You don't want to be squeezed too hard.

MAJ. NEUBECK: Right.

COL. HILL: Dr. Goodman, I appreciate the fact that you're close to verifying what you have suspected for a while and I think it's a great piece of work. I have a program question though. Is the BRAG valve in the F-22 and perhaps Eurofighter programmable enough to make changes at this late date? Of course with G we're dealing with a 10 to 1 ratio, but are we at a stage where we can still make changes to the altitude pressure ratios?

MS MCGARVEY: Yes and no. It's not programmable. However, we are going to reduce the ratio to 3.5 or 3.2 to 1.

COL. HILL: Okay, good.

MS. MCGARVEY: The contractor's breadboard valve was actually performing at about a 3.2 to 1 ratio at preliminary design review.

COL. HILL: Serendipity?

MS. MCGARVEY: It was just the finding.

DR. GOODMAN: I think that pressure ratio would be satisfactory. As a subject in the study, I found that there is a significant pain threshold difference between 3-to-1 and 4-to-1 ratios.

MS. MCGARVEY: We were informed that the contractor was having problems meeting the 4-to-1 ratio, and based on conversations with Maj. Caulkins and literature that Lt. Col. Demitry had available, we decided that we would back off the initial 4-to-1 ratio. The contractor indicated that the valve was performing in the 3.2 to 3.5 range and we approved the lower ratio.

DR. GOODMAN: You will get equal cardiovascular protection at either of the ratios.

DR. ACKLES: It's relatively simple for the manufacturer to change that ratio. It's not difficult to change the redesign of the whole regulator.

MS. MCGARVEY: It will be easier to reduce than increase the ratio. So as we move to production, there may be opportunities such as this that we can implement into the program.

PROF. ERNSTING: The change has already been made in the Eurofighter.

COL. HILL: What pressure ratio are you using?

PROF. ERNSTING: 2 to 1.

PROF. ERNSTING: 2 to 1.

MAJ. NEUBECK: Dr. Goodman, I can't really answer your question whether I will be able to remain at 60,000 feet following loss of pressure in the F-22. I don't know what instructions the technical order will establish for pilot response.

MS. MCGARVEY: The fighting community needs to know the risks of remaining in an unpressurized aircraft at 60,000 feet. Is the risk of hypoxia and DCS sufficiently severe to cause loss of consciousness? If they're in combat and everybody is down below them, they're going to want to stay high and get out of the fray as quickly as they possibly can. We need to give them the probability and severity of the physiologic risk so that they can develop their own procedures for leaving the scene or continuing the engagement.

DR. GOODMAN: The study that needs to be conducted involves risk to the subjects and will need operational support to receive human use approval. It might require: some exposure to lower cabin altitudes on either airmix or 93% oxygen, a rapid decompression through a 5 PSI differential to 60,000 feet, a 5-10 minute period of pressure breathing at 60,000 feet and, descent to 25,000 or 35,000 feet for a selected period of time. It's a risky study, but the question begs for an answer. How long can you pressure breathe with the right cardiovascular support but still escape without getting a DCS hit?

MS. MCGARVEY: It's also going to be important to use an OBOGS oxygen delivery system. All our aircraft are moving to OBOGS systems, so to look at things in 100% mode is not a realistic situation for any of these planes. We need to know the implication of these 93-94% oxygen systems.

DR. GOODMAN: Some of our protocols have been designed with OBOGS systems in mind. Dr. Ackles will probably address those studies. The 88 mm Hg pressure breathing level we studied was chosen in consideration of an OBOGS system.

MAJ. NEUBECK: More than likely, if you tell the pilot he can stay for 10 minutes at an unpressurized altitude of 60,000 feet, he will use the time for egress. He's probably going to leave the threat area, get to the safest place he can find, and then he'll probably descend without having to react to enemy threats at that point. You can cover a lot of ground in 10 minutes at MACH 2, if you have the fuel.

DR. GOODMAN: We think you can do it using a 2-to-1 suit/vest ratio with the ATAGS for 10 minutes. However, we need to actually do the studies in the altitude chamber. It is a very gutsy study to do though.

COL. STORK: One thing we haven't discussed is whether it would be safe to leave the aircraft above 50,000 feet.

COL. HILL: We have a potential answer to that; its called a K36 ejection seat. We've worked on it and find results are quite good at high speed and high altitude. However, it won't fit in our aircraft.

The Medical Aspects of Pressure Breathing

Marvin T. Ryles Sqn Ldr, RAF, MC

Introduction

The first section of this paper outlines medical complications that are theoretically possible when positive pressure breathing for altitude protection (PBA) is undertaken, whilst the second section introduces a study which was recently initiated at Brooks Air Force Base. This study investigates the behavior of intraocular pressure with PBA.

Part I: Medical Complications

At cabin altitudes greater than 40,000 feet, breathing 100% oxygen at ambient pressure is insufficient to prevent features of hypoxia. Under these conditions, the intrapulmonary oxygen pressure falls to a critical level and incapacitation rapidly develops. PBA with 100% oxygen is a well-established procedure for protecting against hypoxia at these higher altitudes.

It is accepted that the total absolute intrapulmonary pressure required for adequate protection is 141 mmHg when breathing 100% oxygen, but that lower levels may be tolerated for short periods if suitable counterpressure is applied to the body (1). The physiological disturbances that invariably accompany PBA increase rapidly with increasing levels of PBA, and limit the useful altitude and duration of protection. These disturbances resolve with cessation of the increased breathing pressure, although it may take several hours for complete recovery. For the purposes of this paper, these commonly occurring physiological disturbances are not considered to be 'medical complications,' although it is appreciated that this distinction is arbitrary and overlap may exist.

It must be stated that, although the following conditions are theoretically possible, there have been no documented occurrences to date. However, continued awareness is required, particularly during development of new PBA schedules. The experience with 1 or 2 minute pressure breathing exposures is extensive. For example, there have been thousands of exposures to a pressure of 30 mmHg and, in the Royal Air Force (RAF), more than 1000 aircrew have received training exposures to 70 mmHg, and this training continues. Longer exposures to pressure breathing have largely been restricted to the research environment, but there are significant examples from sources such as the RAF and DCIEM (Toronto).

Pneumothorax

An increase in transthoracic pressure has the potential to disrupt areas of the lung, especially the terminal bronchioles and the alveolar septae. A static transthoracic pressure of 80 to 100 mmHg is the maximum commonly accepted to be safe in the unsupported chest (7, 11). It is considered that an increase in lung volume is more dangerous than an increase in intrapulmonary pressure and that transthoracic pressures of up to 190 mmHg may be tolerated if the chest and abdomen are supported (11), although direct evidence in support of this is scant. During PBA at pressures greater than 30 mmHg, it is usual to employ chest counterpressure, which has the effect of limiting chest expansion. However, pneumothorax is possible with relatively low lung volumes. It may be that shear forces are generated within pulmonary tissues of heterogeneous compliance, such as between alveoli and blood vessels.

Pneumomediastinum

This condition may occur with relatively little trauma and has even been detected on CT scan in a patient following a simple deep inspiration (2). Under conditions of constant ambient pressure, pneumomediastinum is likely to remain asymptomatic but could be unmasked by a reduction in ambient pressure, and present with features such as a hoarse voice, a sense of fullness in the chest and mild dysphagia.

Surgical (subcutaneous) Emphysema

Gas from a pulmonary defect may track between tissue planes and become apparent as surgical emphysema, easily recognizable by the characteristic feel of subcutaneous crepitus on palpation. Surgical emphysema resulting from pulmonary barotrauma is believed to track to the subcutaneous tissues via the mediastinum, and therefore also suggests the presence of a pneumomediastinum. It is most likely to affect the anterior triangle of the neck.

Arterial Gas Embolism

Arterial gas embolism is a well known, and feared, complication of diving accidents in which lung overpressurization has occurred. The most likely origin of such an embolus is that of gas entering the pulmonary venous circulation via a pulmonary defect. However, no lung damage is clinically detectable in the majority of cases and, although pneumothorax is a common accompanying feature, the absence of a radiologically detectable pneumothorax does not exclude the diagnosis of arterial gas embolism. Pulmonary air trapping is a risk factor for embolism, and therefore conditions such as asthma, lung cysts and pulmonary fibrosis have traditionally been considered to predispose, although there is now considerable debate about the significance of the risk in asthma. Arterial gas embolism in divers has been recorded during dives as shallow as 4 feet. Even though this appears very small, the excess pressure at this depth, when compared to surface pressure, is 90 - 95 mmHg. This figure is broadly in agreement with that quoted as the limit of safe transpulmonary pressure in aviators when the chest is unsupported (7, 11).

Symptoms of arterial gas embolism depend on the target organ. The cerebral cortex is the usual site of clinical involvement and features typically include loss of consciousness, convulsions, hemiparesis, monoparesis, visual disturbance, dizziness and speech difficulty.

Hypertension

Positive pressure breathing increases systemic arterial pressure because of the direct transmission of intrapleural pressure to the left ventricle and to the systemic arteries that are contained within the thorax and abdomen. With sufficient counterpressure, increase in arterial blood pressure may even exceed the level of applied PBA, and arterial blood pressure levels that would ordinarily give cause for concern may be measured. However, as intrathoracic pressure rises in tandem with the applied PBA and increase in intraabdominal pressure almost equals the rise in blood pressure, transmural pressure gradients across vessel walls at these sites are largely unaffected. Likewise, rise in intracranial pressure has the same protective effect.

Areas unprotected from an increase in transmural pressure include the skin, subcutaneous tissues and muscle outside the intrathoracic, intraabdominal and intracranial areas. It is mainly the limb vessels that are exposed to these increased gradients, unless the effect is negated by sufficient counterpressure. Pressure in limb muscle does increase, but not nearly to the same degree as does arterial pressure, unless the muscle is contained within a relatively indistensible fascial sheath. Lack of support to the skin may lead to the appearance of petechial hemorrhages, which typically occur over the face, neck, shoulders, upper chest and upper back. Subconjunctival hemorrhages may also be seen. These cutaneous and conjunctival bleeds, which are not uncommon when levels of 60 mmHg PBA or greater are employed, are considered to be benign.

Hearing Damage

a. <u>Eardrum rupture</u>. Once the eustachian tubes are opened by swallowing, the middle ear pressure is equilibrated with that of the breathing gas. Fryer and Wagner, in 1964, noted eardrum petechiae, bulla and meatal skin hemorrhage at a breathing pressure of 107 mmHg. The use of pressurization of the external auditory canal during pressure breathing at 110 mmHg was successfully adopted in subsequent experiments (4).

b. <u>Labyrinthine window rupture</u>. An increase in intracranial pressure, unbalanced by a concomitant increase in middle ear pressure, has the potential to disrupt either of the labyrinthine windows, although the round window appears more vulnerable to damage than does the oval window. The presentation of such damage may include tinnitus, severe vertigo with nystagmus, and nerve deafness. Labyrinthine window rupture is well reported in divers, but it is suggested that higher pressure gradients are required for fistula formation than are encountered during PBA. In cats, round window rupture occurs when CSF pressures have increased by more than 120 mmHg (6). However, fistula formation does occur apparently spontaneously in the general population and has been suspected in some military aviators exposed to routine cabin pressure changes.

Likewise, intralabyrinthine membrane breaks may also be possible. The effect of such a break would most likely be moderate nerve deafness, with 'notch' hearing loss at one or two frequencies tested at audiometry.

Raised Intraocular Pressure
a. <u>Retinal hemorrhage</u>: An increase in intraocular pressure during pressure breathing would minimize retinal vessel transmural pressure gradients and would thereby help to protect against retinal hemorrhage. Green (1961) investigated the effect of pressure breathing on human retinal vessel diameter and noted that there was no increase in diameter during short duration exposures with levels of up to 75 mmHg. It was argued that this lack of increase in vessel size indicated that there had been no significant distention of the outer coats of the eyeball. By implication, this would suggest an increase in intraocular pressure (5). In 1967, Segal used tonometry to measure the intraocular pressure during pressure breathing of 14.6 and 29.2 mmHg for up to 4 minutes. There was an overall increase in intraocular pressure, but the response differed considerably between subjects (12).

b. <u>Retinal vein occlusion</u>: An association between retinal vein occlusion and raised intraocular pressure is recognized in clinical practice. However, during PBA, the situation is physiologically different in that blood pressure is also increased; the time during which intraocular pressure alone is raised is the period between cessation of PBA and normalization of intraocular pressure. There is one case of retinal vein thrombosis that is associated with pressure breathing that, although unpublished, is well known to many people who work in altitude protection. In 1988 at Brooks Air Force Base, a subject developed a left sided central retinal vein occlusion 24 to 48 hours following an experimental exposure to simulated altitude. The exposure had included a rapid decompression from 20,000 feet to 50,000 feet in 1.5 seconds, followed by 30 mmHg of pressure breathing for one minute prior to descent to lower altitude. Following resolution of the venous occlusion, the subject was medically cleared to resume exposure to simulated altitude, but was advised to avoid rapid decompressions and positive pressure breathing. Although it is not possible to exclude a causative role of pressure breathing, it is considered unlikely. The symptomatic presentation of retinal venous occlusion may be delayed by 24 to 48 hours, but is unusual. The exposure was to 30 mmHg, a low level at which a lot of experience has been gained. Also, retinal vein thrombosis does occur apparently spontaneously in the general population, although it does tend to present at an older age than this particular subject.

c. <u>Closure of the angle in a narrow anterior chamber</u>: It is possible that an increase of intraocular pressure may close a shallow anterior chamber, thereby precipitating closed angle glaucoma.

Long Term Exposure
There have been no documented reports of adverse long-term effects of exposure to PBA. At the 1994 ASMA conference, the findings of the annual medical examinations carried out on a group of 14 experimental subjects at DCIEM were presented (3). These subjects had been repeatedly exposed to levels of PBA up to and including 88 mmHg over the course of 3 years. The examinations, which included stress ECGs, chest X-rays, pulmonary function testing, echocardiography, and blood and urine analysis revealed no pathology.

Medical Complications—Conclusion

In summary, although the conditions outlined above may theoretically occur, there is no documented evidence to support their occurrence at the levels and durations of PBA employed to date. Provided that adequate

counterpressure is used and suitable training is provided, it appears that the risk of developing serious medical complications is low. Certainly, at 30 mmHg of breathing pressure, where there has been experience with thousands of individuals over the years, the risk of serious medical complication seems vanishingly small. PBA at levels up to 88 mmHg, or at lower levels but for time periods extending beyond the traditional 1 to 2 minutes, again appear safe. However, the number of exposures under these latter conditions are limited, warranting continued awareness.

Part II: The Effect of PBA on Intraocular Pressure

Introduction
Historically, the observation that petechial and subconjunctival hemorrhage occurred during pressure breathing led to concern regarding the possibility of retinal hemorrhage. However, it was hypothesized that an increase in intraocular pressure occurring at the same time as the increase in vascular pressure would lessen any increase in retinal vessel transmural pressure, and so protect. What constitutes a safe transmural gradient is unknown, although Henry in 1950 suggested a maximum of 50 mmHg, albeit in the context of negative G_z acceleration (8).

Because of interest in the effects of PBA, a study has been initiated at Brooks AFB to investigate the following:

1. The intraocular pressure during exposure to PBA levels of up to and including 60 mmHg for periods of up to 10 minutes. This will provide information on intraocular pressure behavior with levels and durations of PBA that are currently of interest.

2. The intraocular pressure following cessation of the above PBA levels. It is generally believed that intraocular pressure falls promptly once PBA is stopped. This is logical because of the relationship between intraocular pressure and ocular venous pressure (10), but direct evidence is lacking. If the intraocular pressure were to remain raised for a prolonged period of time once systolic blood pressure had normalized post pressure breathing, it is possible that retinal damage could occur.

The study is at an early stage, but the following outlines the framework of the protocol and tentatively presents the findings to date.

Method

It is intended to use 15 subjects of either gender; 4 subjects have now completed the protocol. Counterpressure is worn at all pressure breathing levels and consists of a CSU-17/P thoracic garment together with ATAGS (Advanced Technology Anti-G Suit). The ATAGS provides counterpressure to approximately 90% of the surface of the lower limbs and the pelvic area. In addition, the standard Combat Edge helmet (HGU-55/P) and oro-facial mask (MBU-20/P) are worn. The helmet contains an occipital bladder, which inflates during PBA to increase mask tension and so aid mask-to-face sealing. The ratio between breathing pressure, thoracic counterpressure garment and the G-suit used in all experimental exposures is 1:1:3.

After initial equipment fitting, subjects receive training in PBA until both 30 and 40 mmHg can be reasonably tolerated for 10 minutes and 50 and 60 mmHg have both been experienced for several minutes. As an aid to reducing hyperventilation, subjects are able to see their end-tidal carbon dioxide level in real-time, and attempt to prevent the value dropping below 30 mmHg.

Prior to the first training PBA exposure, and following the last data collection PBA exposure, the subjects attend an ophthalmological examination at the Consult Service, Brooks Air Force Base. The examination includes corrected and uncorrected visual acuities, intraocular pressure measurements, slit lamp examination, fundoscopy, detailed visual field mapping and the taking of retinal photographs.

The levels of PBA employed are 30, 40, 50 and 60 mmHg, which encompasses levels presently used in the Combat EDGE system. The exposure to each level of PBA is 10 minutes, unless limited by subject fatigue or other factors. Intraocular pressure is recorded in the right eye before each exposure, at intervals of 1 to 2 minutes throughout the exposures, and at 1 minute intervals after the exposures until intraocular pressures have normalized. Intraocular pressure values are obtained by using the Tono-Pen XL Tonometer, whose accuracy has been demonstrated to be acceptable when compared to that of Goldmann applanation tonometry which is, itself, generally accepted to be the most accurate non-invasive technique (9). The Tono-Pen is a contact device and therefore requires the use of a topical anaesthetic. The pressure readings are taken by staff from the Ophthalmological Consult Service, Brooks Air Force Base.

In addition to intraocular pressure, other parameters such as heart-level arterial BP, are recorded.

Observations to Date

It is too early to produce conclusive results as only 4 subjects have completed the protocol to date; however, the responses of these first subjects are illustrated below.

Intraocular pressure

Data from the first few subjects support the belief that intraocular pressure rises with PBA (Figures 1 to 4) and, in a given individual, the greater the applied breathing pressure, the greater the increase. However, as observed by Segal (12), there is marked individual variation. It appears that, following the initial increase at the onset of PBA, the level of intraocular pressure was broadly maintained i.e., there was no discernible trend for either a decrease or a continued increase in pressure over time.

Following cessation of PBA, intraocular pressure levels typically returned to baseline within 2 to 6 minutes, although 14 minutes elapsed before normalization in one subject who had been exposed to 40 mmHg for 10 minutes (Figure 5).

Ophthalmology Examination

There have been no changes detected between the pre- and the post-ophthalmological examinations. These particular examinations were performed within a few hours of completion of the 60 mmHg exposures.

General Features

a. Many of the cardiovascular features that traditionally accompany PBA were not present. This was probably because of the combination of the extended coverage provided by ATAGS and the use of the 1:1:3 breathing pressure: chest counterpressure: G-trouser ratio, leading to improved cardiovascular support. Pulse and pulse pressure changes were not apparent when compared before, during and after the exposures. However, as anticipated, there was an increase in both systolic and diastolic arterial BP during exposure to PBA.
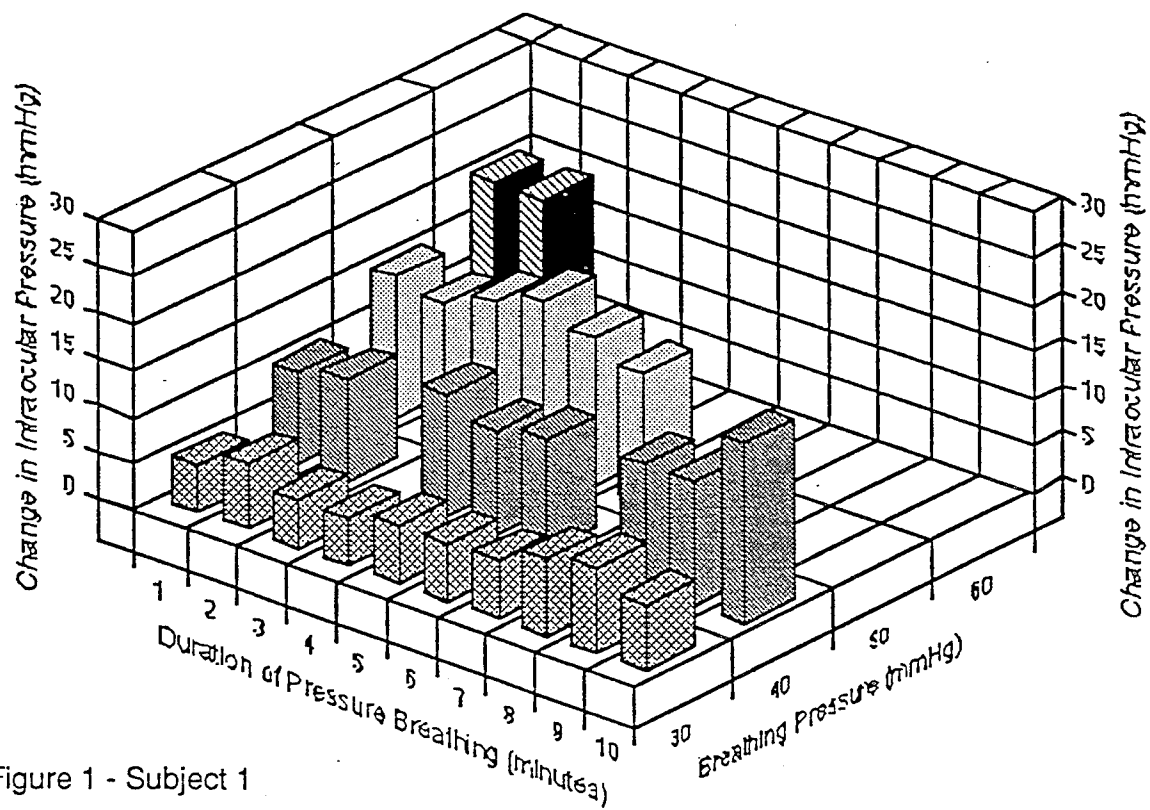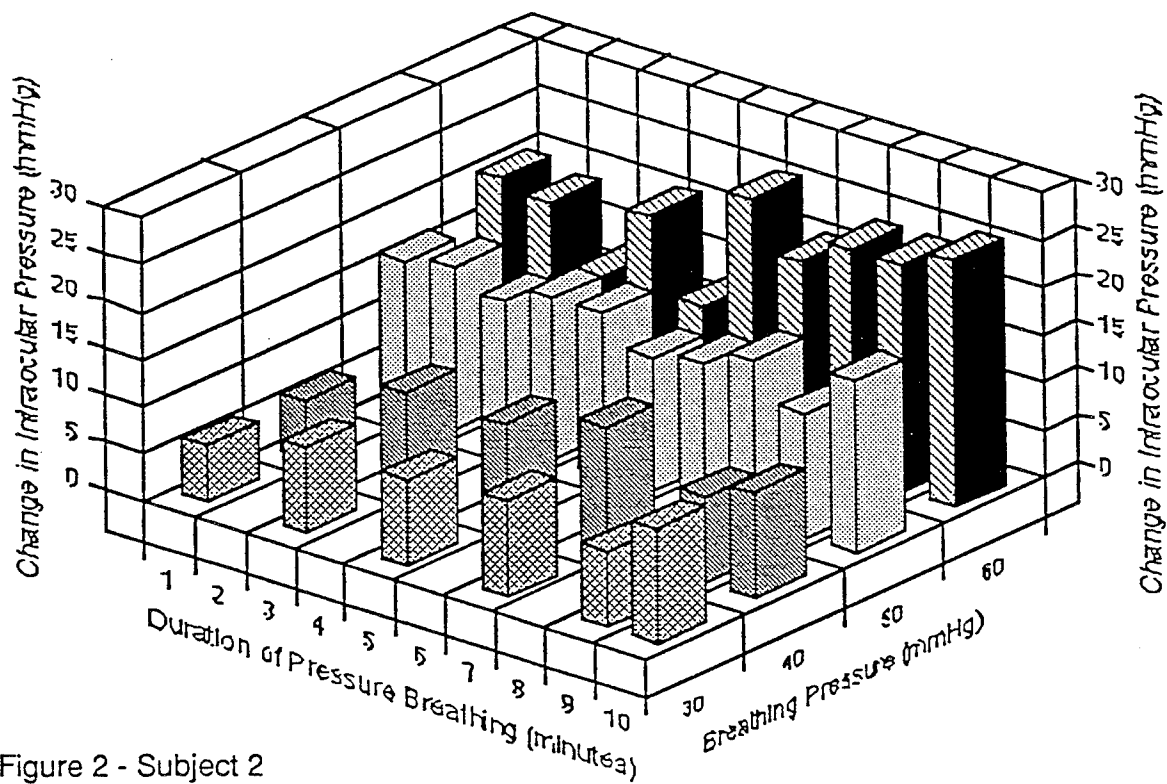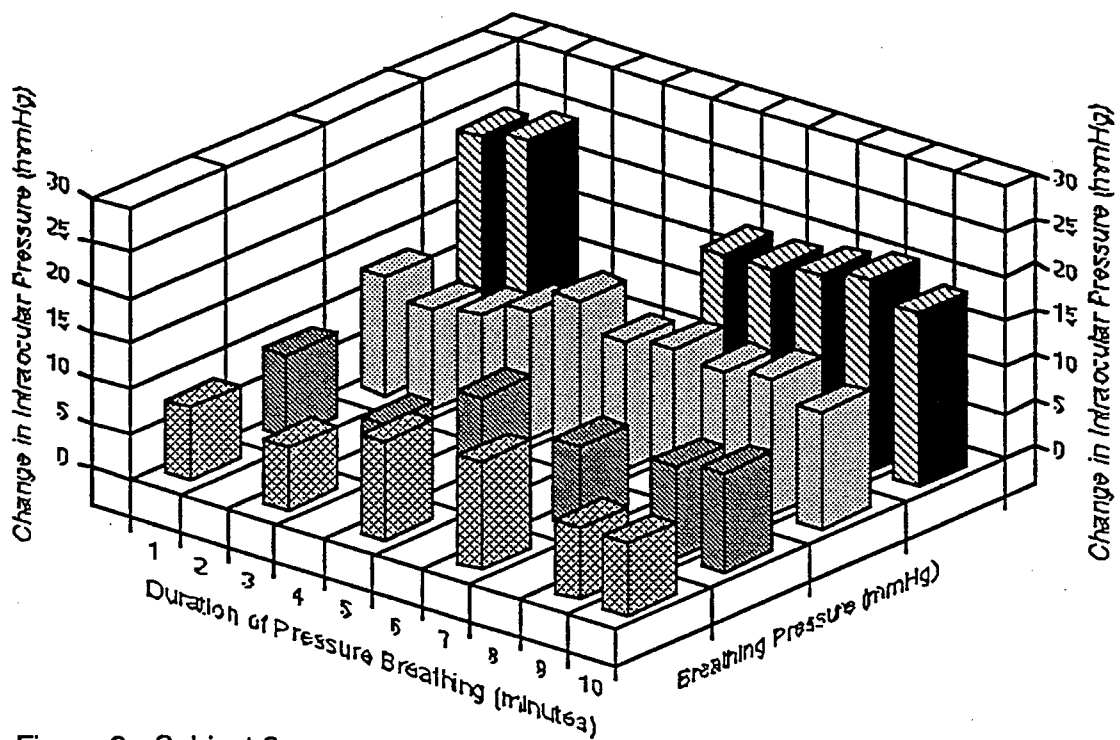
Figure 1 - Subject 1

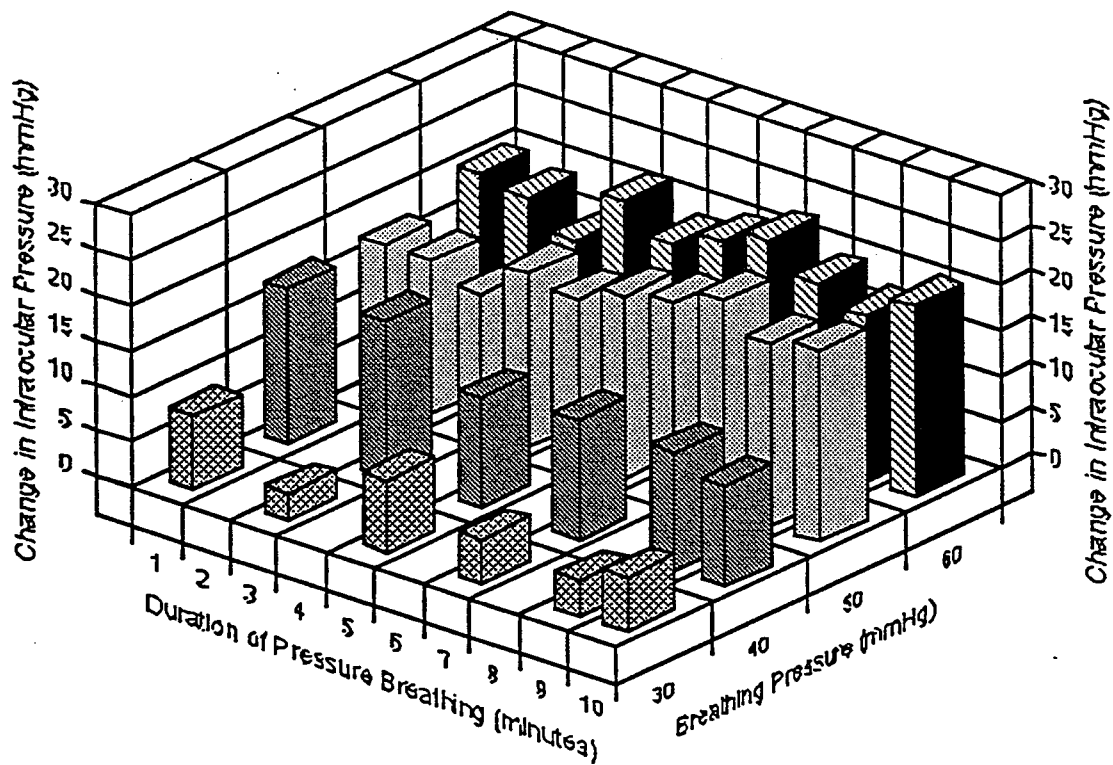Figure 2 - Subject 2

Figure 3 - Subject 3

Figure 4 - Subject 4
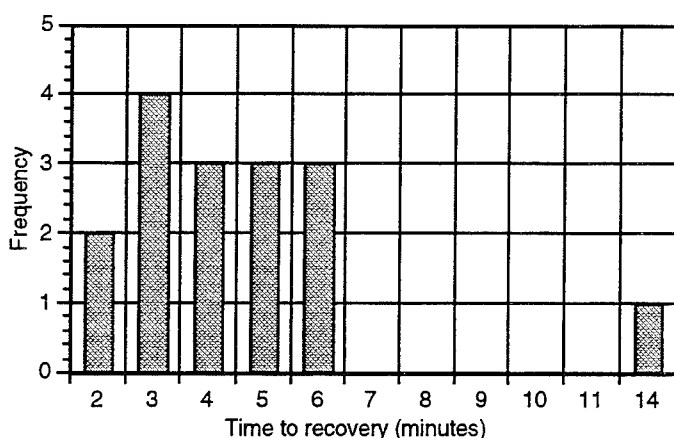
Figure 5. Recovery time of intraocular pressure after cessation of positive pressure breathing.

b. A well recognized effect of PBA is the almost invariable occurrence of significant hyperventilation. It is believed that the use in this study of visual feedback of the expired carbon dioxide partial pressure aided training, enabling subjects to maintain an expired carbon dioxide pressure of between 30 and 40 mmHg.

c. It was felt by the subjects to date that the main limiting factor to PBA at 50 and 60 mmHg was neck discomfort. There were no reports of pre-syncopal symptoms.

d. Petechial hemorrhages occurred in 3 subjects when exposed to a breathing pressure of 60 mmHg. A subconjunctival hemorrhage occurred in one subject when exposed to 60 mmHg PBA.

e. The life-support equipment worked well, with minimal mask leakage problems and no excessive discomfort during garment inflation. No more than mild, transient pins and needles occurred following suit deflation after any exposure. Greater difficulty with mask sealing at the higher levels of PBA may be encountered in the field if careful attention is not paid sizing and firmness of fit.

The Effect of PBA on Intraocular Pressure--Conclusion

The observations to date indicate that intraocular pressure does increase with increased breathing pressure, although not on a one-to-one basis. At levels of PBA of up to and including 60 mmHg, it seems likely that the degree of increase of intraocular pressure was sufficient in our subjects to ensure that the increase of retinal vessel transmural pressure was less than the 50 mmHg limit suggested by Henry (8). There was no apparent trend for decrease, or further increase, in intraocular pressure during continued PBA exposure and, upon cessation of PBA, the intraocular pressure usually returned to baseline levels within 2 to 6 minutes.

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Modeling PBA Gas Dynamics

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1.0 Introduction

Mathematical modeling and simulation have become the norm in modern science as tools for analysis of complex problems. Mathematical models are popular because of the very useful way they serve to isolate a particular aspect of a complex system and to dissect it into easily understandable parts. They are valuable for encapsulating existing knowledge and exposing ignorance by making the unknown explicit. The aim of this paper is to review the current state of modeling as it relates to describing the physiologic performance breathing systems employed in high-performance military aircraft. Specifically reviewed are the background and progress toward development of a dynamic gas exchange model of the system comprising the breathing system and the cardiopulmonary system of the aviator in the PBG/PBA environments.

2.0 Background

Models of steady state respiratory gas exchange are well established[1]. The most commonly employed of these is the Alveolar Gas Equation (AGE)[2] that describes the steady state relationship between barometric pressure and the fraction of oxygen and carbon dioxide in the alveoli. Although useful in many aerospace applications, the AGE cannot describe the breath-to-breath transients that occur during ordinary breathing, nor can it be employed to describe the dynamics of respiratory gas exchange during rapid changes in ambient pressure or when there is a net exchange of nitrogen between the lung and the environment. During most flight operations, even at high altitudes, as long as the aircraft cabin pressurization system remains functional, the AGE may be employed to set the breathing system design criteria for avoiding undue hypoxia. However, during rapid changes in cabin altitude and/or changes in the pressure or composition of the inspired breathing gas, the assumptions required for use of the AGE are violated. Thus, there will be significant errors between the actual alveolar gas composition and that predicted by the AGE until the respiratory steady state is re-established.

To adequately describe respiratory gas dynamics and the pressure/composition transients accompanying breathing, a more comprehensive model of respiratory gas dynamics is required.

2.1 Dynamic Model of the Aviator's Breathing System

Recently, the USAF Armstrong Laboratory sponsored a feasibility study by Biodynamic Research Corporation to investigate the possibility of creating a high-fidelity model of the gas dynamics in the Aviator's Breathing System (ABS) shown in Figure 1. The study laid the foundation for creating an integrated mathematical model of the ABS for validation and subsequent use by the USAF as a research tool in their PBG and PBA life-support system research and development programs. The following paragraphs describe the background, findings and recommendations from the AL/BRC study. The complete report is available from the USAF Armstrong Laboratory.[3]

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Aircraft O₂ System

Breathing Regulator

$\bar{Q}_I = \dot{V}_I$ $\bar{Q}_E = \dot{V}_E$

Air O_2

Cardiopulmonary System

H_2O V_L

Lung

O_2 N_2 CO_2

Pulmonary Circulation

Q_S

LA

RA RV -----Heart----- LV Q_T

Q_T

O_2 N_2 CO_2

O_2 N_2 CO_2

Systemic Circulation

©BRC
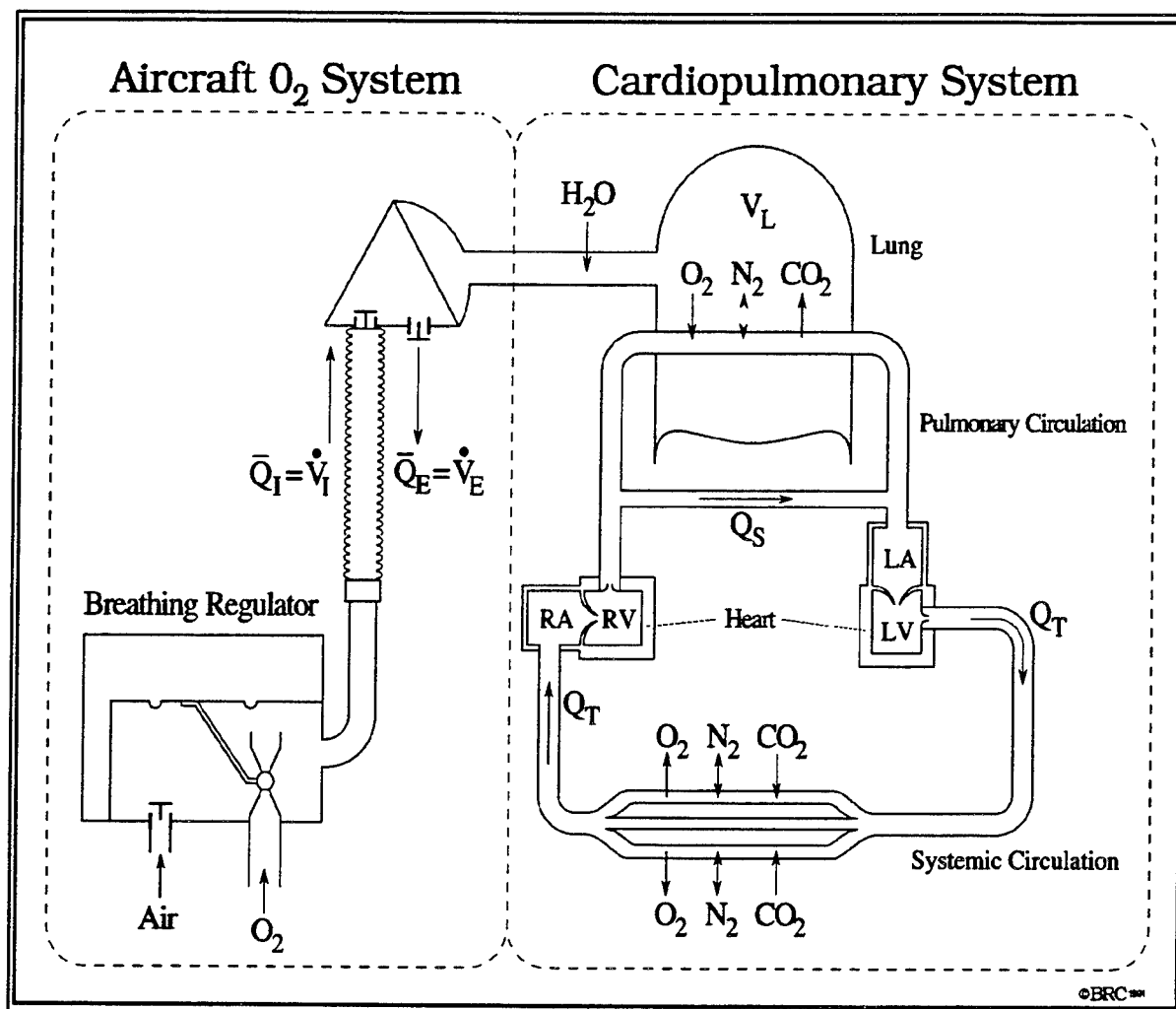
Figure 1. Aviator's Breathing System.

The feasibility study concentrated on developing computer software modules that were capable of simulating the main effects of environmental variables on the breathing system hardware and respiratory and cardiovascular systems. Software modules were developed and tested for simulation of: (1) the flows and pressures within the breathing gas delivery system; (2) the flows, pressures, and respiratory gas distribution within the lung; and (3) the steady-state flows and pressures within the cardiovascular system. Subprograms were also developed to compute altitude-barometric pressure relationships as well as passenger cabin pressures in military aircraft.

In addition to the software development, BRC reviewed and organized the Government-furnished data from a series of manned rapid decompressions known as the "EONS Experiments." The data from approximately 170 experimental decompressions were screened for their suitability for use in parameter selection and validation of the respiratory modeling software. The data appears to be highly coherent and fully usable for model validation. The summarized results of the EONS Experiments represent a new physiologic database that is directly applicable to USAF operational scenarios. The respiratory gas composition time histories represent original scientific data suitable for publication and use by the aerospace medicine community independently of their value in the ABS modeling effort.

The following paragraphs briefly describe some of the technical aspects of the prototype ABS model modules developed during the AL/BRC feasibility study. The complete model and the EONS data are described in the Final Report.[3]

2.2 The ABS Oxygen Delivery System Model

There are ample laboratory data describing the flow/pressure relationships within breathing systems.[4,5,6,7,8,9,10] However, other than the AL/BRC study, the author is not aware of any attempts to model USAF breathing gas delivery systems. There is one report that describes a model of a simple US Navy demand regulator, but no details of the system equations were given.[11] The breathing system model created by BRC simulates the pressures and flows in a generic oronasal mask during breathing through a diluter-demand breathing regulator.

Because breathing regulators are inherently nonlinear devices, deriving and solving models of their function is difficult. One alternative method of modeling regulators is to characterize their performance parametrically over the operating range of interest. Except for the Navy study cited above, parametric modeling of empirical data has invariably been used for describing regulator performance. A more rigorous, but much more difficult method is to write and solve the system equations for the regulator of interest. For the ABS model, BRC modeled the regulator performance parametrically, while the oronasal mask was modeled by solving the system equations describing its performance. Both models were created so that their performance was consistent with the requirements of Air Standards issued by the Air Standardization Coordinating Committee.

2.3 The ABS Respiratory Function Model

To adequately describe breath-by-breath gas dynamics in the lung during pressure/composition transients, a model that includes the spatial and temporal variation of ventilation and perfusion within the lung is required. Several models are described in the literature that describe cardiopulmonary function in clinically significant pulmonary disease or for teaching respiratory physiology.[12,13,14,15,16,17,18] Unfortunately, these were developed for modeling a particular physiologic response or for a particular environment or clinical situation. For the most part, previous models are not easily adaptable to the high-performance aviation scenario because they do not describe many of the relationships between the environment, the breathing system, and the crew member's other protective systems. As an alternative, BRC proposed the development of an ABS model tailored to model the gas flows and composition within the breathing and respiratory systems in typical operational and experimental environments encountered by military aircrews.

For the ABS feasibility study, two modules were developed that together describe the vertical ventilation pattern in the lungs and the concentration of a tracer gas throughout the lungs during inspiration and expiration. The lung is modeled as four regions of equal mass that are located along the vertical axis of the lung. The model accounts for the volume and ventilation differences created by gravity, which have traditionally been called "interregional differences" by physiologists. A detailed description of the ABS models is given in the final report[3].

2.4 Modeling the Cardiovascular System

Ultimately, the ABS model seeks to describe the cardiopulmonary physiology associated with the various environmental stressors associated with high-performance flying. Early in the feasibility study, it became apparent that a reasonably complete model of the cardiovascular system would be required to adequately model the effects of positive pressure breathing (PPB) for altitude and acceleration protection (PBA and PBG). Thus, it was decided to create a steady-state cardiovascular model for the prototype program that could be extended to a dynamic model in the integrated ABS model. BRC concentrated the literature search on modern cardiovascular modeling work that appeared to be sufficiently comprehensive, but not overly complicated. In particular the cardiovascular model for the ABS paralleled the developments presented in a paper by White, et al.[19] It also draws from papers by Jaron, et al.,[20,21,22] whose approach was similar to that presented in earlier papers by Rideout,[23] Snyder,[24] and Avula.[25] All of these authors cited the earlier work of Womersley[26] who developed equations describing the convection of fluids in elastic tubes, as well as those describing the propagation of pressure waves and pressure dependent changes in tube radius.

2.5 Integrated Models of Cardiopulmonary Function

The author is aware of only one integrated model of the physiologic function of the human body, a computer model known as Human[27]. Human is an integrated computer model of the entire body that is used as a teaching program for medical students. BRC reviewed Human during the feasibility study and was impressed with its fidelity, given its simplicity.

The general approach employed in Human is to model physiologic function by use of curve fits to empirical data. This approach limits Human's use to conditions that have been incorporated in the model. Unfortunately, Human as presently configured only allows small excursions from sea level, +1 Gz conditions. However, the approach employed in Human to implement physiologic control systems, such as the baroreceptor system for control of cardiac output, is simple and effective. It would be relatively straightforward to refine and modify the Human code to simulate the range of conditions required for the ABS model.

2.6 Modeling the Environment

The physical environment includes such things as the barometric pressure, the temperature, the acceleration, etc. Most environmental variables were implemented as inputs to the hardware and physiologic models. The prototype ABS Model includes subroutines that model the variation in atmospheric pressure with altitude as well as the change in MILSPEC cabin pressure with altitude.

2.7 Summary of the ABS Feasibility Study

At the present time, there are no comprehensive physiologic models that can describe the physiologic consequences of changes in operating conditions and design of the Life-Support Systems (LSS) employed in modern high-performance aircraft. BRC has created tentative computer models of the components required to implement an integrated model of the ABS that simulates the function of the hardware and the physiologic subsystems in response to environmental changes. The Final Report[3] gives detailed descriptions of the newly developed models. Summaries are given below with comments on the improvements necessary to create an integrated ABS Model in a follow-on development effort.

3.0 Findings and Recommendations

The work accomplished in the AL/BRC feasibility study has laid the groundwork for developing an integrated ABS Model. BRC wrote prototype software modules that form the major building blocks for the integrated ABS Model. They concluded that the development of an integrated ABS Model was feasible and desirable. There appears to be sufficient extant data for selection of parameters and independent validation of the major components of the ABS model. However, the complete validation of the combined effects of acceleration, pressure breathing and altitude on the ABS must await new experimental data. Fortunately, the attempt to simulate the physiologic response to combined stressors will explicitly point to missing data and guide the development of experimental protocols to elucidate their significance. The following section outlines the major steps required to create the fully integrated ABS model by building on the work accomplished in the feasibility study.

At present, none of the prototype ABS modules is fully validated. Therefore, prior to their integration, additional development of the prototype software modules will be required to ensure their ability to independently simulate the physiologic response to changes in their input variables. In addition, the software code and numerical methods for solution of the system equations will be optimized and, where possible, standardized across modules. This will result in more efficient code and ease the process of creating the software interfaces necessary to integrate the software modules into a fully integrated ABS Model. The following sections outline the major work on each software module required before their integration.

3.1 Breathing Gas Delivery System

The breathing gas delivery system is comprised of a source of breathing gas, such as an oxygen supply or a Molecular Sieve Oxygen Concentrator (MSOC), a demand regulator that regulates the flow and pressure of the breathing gas in response to the changes in the ambient pressure and acceleration, and finally an oronasal mask that ensures unidirectional flow of breathing gas and forms a seal to the face of the aviator.

The preferred way to model the breathing gas source would be to model the composition at the demand regulator inlet as a function of the mass flow drawn by the regulator and the ambient pressures. This approach will work for both MSOC-based systems or systems based on storage of gaseous or liquid oxygen. Backup oxygen systems such as seat-mounted Emergency Oxygen Systems are relatively simple to model. Regulated Back-Up Oxygen Systems, such as those employed with MSOCS can be modeled similarly to the demand regulator, but without dilution of the feed gas.

The demand breathing gas regulator in the present model assumes the flow demanded is supplied at a pressure typical of the functional performance of an ASCC demand regulator. For the ABS regulator model, it will be necessary to completely parameterize the mass flow-pressure characteristics of the regulator and include G-sensitivity as required. Parameterization of the regulator's performance is preferable to any attempt to model the internal hardware function. Breathing gas regulators are highly nonlinear devices and the effort required to write and solve the equations describing the flow-pressure characteristics of even a simple regulator would be substantial. The regulator model will also include a parametric representation the regulator's supply gas dilution characteristics as function of ambient and outlet pressures. The regulator outlet relief valve will be modeled on the characteristics of a poppit relief valve rather than the ideal relief included in the present model. The mask tube and connector flow-pressure characteristics will employ the isothermal tube flow equations and will allow for turbulent pressure losses.

The oronasal mask model is essentially complete as created in the feasibility study. The model is based on the functional performance of an ASCC compliant breathing mask. The present model can be modified to change the mask valve area versus pressure function to match an actual mask if desired. Empirical data can be employed to match the valve characteristics in the model to actual mask flow-pressure data.

3.2 Pulmonary Model

The ventilation pattern in the Pulmonary Model is presently driven by the flows at the mouth. In order to integrate this model with the mask/regulator model, it will be necessary to make pleural and ambient pressures drive the gas flows in the model, i.e., the pressure difference across the chest wall. This will require adding the elastic characteristics of the chest wall to the Pulmonary Model and changing the logic of the numerical solution scheme. This addition will make the model more realistic as pleural pressure changes normally drive gas flow, and in the case of a rapid decompression, changes in the ambient pressure, coupled with the pleural pressure, and airway resistances creates gas flow in the lungs.

Presently the model assumes that the ambient pressure remains 1 atm and that the density of the gas in the lungs remains constant. Obviously, this is not the case when pressure breathing and rapid decompressions are modeled, so the Pulmonary Model will require modification to include the density effects of lung gases that are not presently included. This can be done with relatively little difficulty.

The Pulmonary Model inherently contains the effect of acceleration on ventilation through the G-terms included in the system equations, but the resulting regional volumes and flow patterns for G-values greater than +1 G_z have not been studied in detail, and the performance of the model in predicting ventilation patterns in high-G environments will require verification.

In order to integrate the Pulmonary Model with the Cardiovascular Model, the transport of carbon dioxide and oxygen between the alveolar gas and the blood must be included. The model presently tracks the concentration of a single gas in the airways of the four regions, therefore it cannot realistically include the effects of more than one tracer gas species. To accommodate alveolar exchange, the model must be modified to trace multiple gas species and to include the transport of oxygen, nitrogen, and carbon dioxide throughout the airways and alveoli of each region in the model.

3.3 Cardiovascular Model

The major change required in the cardiovascular model will be to incorporate dynamic response characteristics and add models of the short-term physiologic control systems. The steady-state effects of acceleration are already implemented in the cardiovascular, but the modifications will be required to include transient changes in flow pressure within the segments. Pressure breathing effects will be implemented by modeling the rise in intrathoracic pressure and its effect on venous return and blood pressure. Methods of modeling pressure garments such as partial-pressure garments and anti-G suits must also be added. It should be feasible to model the application of external pressure by manipulation of the tissue pressure that is already included in the cardiovascular model. The current version of the model also runs very slowly with the Runge-Kutta numerical integration algorithm presently employed. Different methods of formulation and solution of the differential equations should be investigated to optimize the speed and stability of the model.

The pulmonary cardiovascular section will be split into four subsections each supplying the corresponding region of the pulmonary model. Local regulation could be simulated as will flow pressure alterations in response to acceleration and pressure breathing.

3.4 Integration of Subsystem Modules

In the feasibility study, BRC created basic software modules and a framework for an integrated ABS model. As modern life-support systems (LSSs) are substantially integrated, the integrated model should attempt to include the interaction of the relevant LSS components with the environment and the crew member and other parts of the LSS. Most of these variables could be simulated parametrically as inputs to the system to model PBG as well as anti-G suit inflation during pressure breathing for altitude (PBA). Modeling of interfaces will likely be one of

the most interesting and most difficult parts of combining the prototype ABS modules into an integrated ABS model. Just as importantly, with the aid of the ABS Model, it should be possible to discover the nature of improvements that would be necessary to correct poor interaction or poor integration between the crew member and the LSS. The integrated ABS Model could be employed to perform parametric studies as an aid to illustrating and understanding the nature of the interactions between the environment, the LSS, and the aviator. A knowledge of the (dynamic) nature of the interaction between the physical and physiologic subsystems will also help guide the USAF's research program toward elucidating the physiology necessary to describe the effects of LSS design on aircrew protection.

Ideally, the final model will be a single software module with a "user friendly" interface that allows easy selection and adjustment of input variables and display of simulation results. Finally, it is recommended that further development proceed methodically and be spread over a sufficient time to allow for development of new data that may be required. The prototype model modules should be developed as independent modules for use independently of the integrated ABS model. This will permit "stand alone" use of the modules or for their further development for projects outside the context of the ABS.

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Discussion

DR. BOMAR: There are some things that should be kept in mind when you look at the results of any modeling effort. A lot of modelers get caught up in their models and they begin to talk about the model as if it were real and they say, "when we use this input, this is what happens." It should be remembered that the model is not real; data are real. When you see an extrapolation, you should just back off and think very carefully about the numbers. Interpolations are generally more valid, but they are also suspect. Some of Dr. Stolp's work supports my suspicion that the use of end-tidal oxygen to estimate arterial gases is at least suspect and that we really need to do invasive work to understand how to use these data to estimate our arterial gases.

DR. BAUMGARDNER: I would agree with your comment that the Jaron model is probably more complex than you need for cardiovascular modeling. I don't know if you're familiar with some of Mike Hlastala's current work on some deviations from the West hydrostatic model. You might be interested in talking to him about some of the pulmonary blood flow distributions under G which deviate from the West model. I think that would fit very nicely in what you're doing.

DR. BOMAR: The cardiovascular portion was Dr. Scott's work. I'm less familiar with that part of the model.

DR. BAUMGARDNER: It might be worthwhile for you to contact Mike Hlastala. He's been doing some work here in the Armstrong Laboratory in Dr. Burns' Branch and is also doing some work in his laboratory at the University of Washington.

DR. ACKLES: It's too bad Dr. Fraser is not here to discuss their modeling efforts. We have several models running in the laboratory. One of the things they are discussing is modeling the cardiovascular pulmonary model during exposure to high G and altitude. He's been working this problem for several years and has put together a group from the US, Canada and the Navy. I think there is someone from Brooks AFB in the group.

Pulmonary Overpressure

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Introduction

Manned flight in the new generation of fighter and attack aircraft will be physiologically more demanding than any other aircraft in the inventory.[5,34] Restrictions placed upon life-support equipment in order to achieve performance objectives place a premium upon all equipment used, and has forced a re-examination of currently imposed safety limits.[1,72] With increased operational capability, including regular flights to over 50,000 feet projected, pressure suits, and/or cockpit differential pressures greater than 5 psi, (260 mmHg), must be used to support the pilot.[8,21,29,40,52,53] Pulmonary overpressures from decompression events will occur.[56,57,58] In addition, positive pressure breathing for G protection is being further developed, refined, and employed, placing pilots at risk for overpressure incidents.[13,25,55,67,68]

Current pressurization schedules are based on poorly understood human tolerance limits. Static pulmonary pressure limitations are being applied to situations where dynamic pressure limitations are more appropriate. The purpose of this report is to examine the currently available data, and provide a recommendation for current operations. The limit of dynamic pulmonary over-pressurization is to be identified ideally with reference to the population of interest, the current United States military aviator, and is not meant to be applied to the general population in a clinical medicine setting. Ideally, this dynamic pressure limitation should be isolated from the physiologic confounders of high sustained G, hypoxia, and the low pressure effects of decompression sickness and ebullism. However, the human data is incomplete, and recommendations are based in part on extrapolation from animal data. The next section defines the overpressure problem.

The Dynamic Overpressure

The ability of the lungs to tolerate changes in pressure determines guidelines for clinical medicine, diving practices, and aviation practices.[66] In clinical medicine and in aviation medicine, positive pressure breathing equipment applies a positive pressure into the lungs, creating a pressure gradient between the alveolus and the atmosphere.[11,17,22,24,26,35] Usually, a constant pressure is applied to the respiratory system over a period of seconds to hours. During the application of this "static" pressure, there may be transient peaks of "dynamic" pressure greater than intended, which last for fractions of a second to seconds. A dramatic example of a dynamic overpressure situation lasting fractions of a second occurs during the explosive decompression of a fighter cockpit. Decompression in diving or aviation creates a pressure gradient between the alveolus and the atmosphere when the ambient pressure drops below the alveolar pressure. The duration of the pressure gradient caused by decompression is usually on the order of fractions of seconds to several seconds, and is a dynamic pressure, Figure 1.

In order to develop a standard for tolerable dynamic pressure, the conditions must be stipulated. The population for which the limit is defined is the awake and alert military aviator. The air is assumed to be wet, at room temperature. The airway is free from internal and external obstruction. The chest wall is free moving. The phase of respiration at which the decompression occurs is resting at the functional residual capacity, FRC. In the absence of the peak pressure measured at the mouth, the conditions necessary to characterize the decompression are the initial and final pressures and the duration over which the decompression occurred. Pulmonary damage is defined by any objective measure of pulmonary damage, and must be measured in isolation from effects of hypoxia, ebullism and evolved gas. A useful variation on this limit is the supported dynamic pressure. In this case, the aviator is awake, alert, and wearing some life-support equipment. Unfortunately this will vary according to the equipment used. In all instances, variations from these conditions will be stated where appropriate.
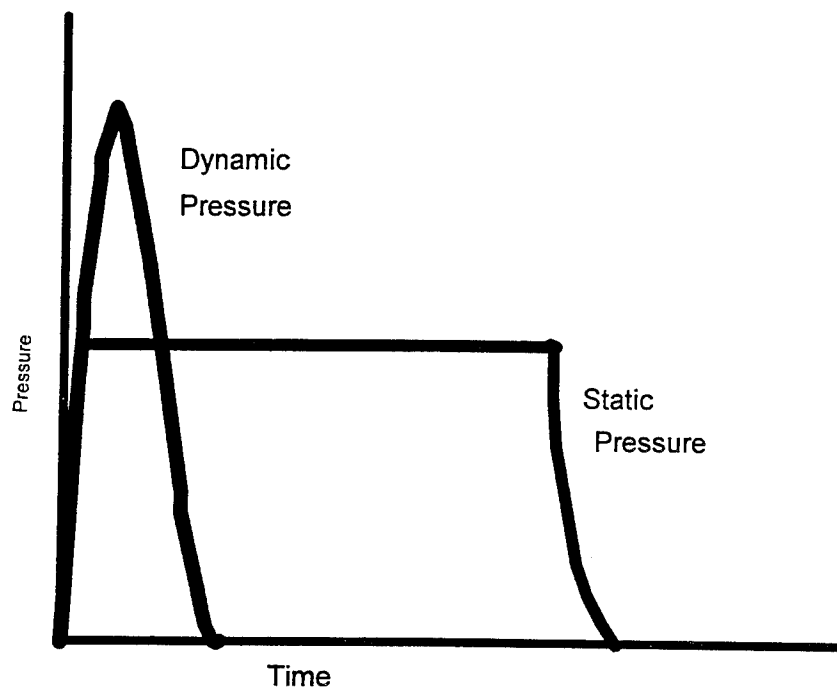
Figure 1. Diagramatic representations of positive dynamic and static pulmonary pressures over time.

The Current Pulmonary Pressure Guidelines

Most readers are familiar with the static limits of 80 mmHg pressure for a human with an unsupported chest wall,[9,53,62] and 190 mmHg for a supported chest wall.[34,50,51,66] The static unsupported chest wall limit has been passed through the literature since the 1930s. Polak and Adams, 1932,[63] are cited often as having written the first paper promoting the 80 mmHg limit for safety. Their paper was written in response to sudden deaths of naval personnel that occurred immediately after ascent in emergency escape training for submarines. It took an original experimental design to demonstrate the air emboli in the carotid circulation of dogs. Pressures of less than 80 mmHg did not usually cause embolism, while pressures greater than 90 mmHg usually caused emboli. This paper established the etiology of air embolism as a pulmonary overpressure and distension phenomena. It did not report experimental results on human subjects, or seek to prove or establish a range of pressures or norm for maximum safe pressures. The authors were often misrepresented as having established a safety limit for decompressions. In a similar manner, Benhke, 1933, is cited.[20] No firm data is presented in his paper either.

Experimental work looking at peak static pressures causing pulmonary pathology has also been cited as substantiation for a dynamic overpressure limit. In the most well known of these studies fresh, unchilled human cadavers were used to determine pulmonary pressure limits. Malhoutra and Wright, in 1961,[60] set out to determine the static pressure limits of the lungs and the effects of binding the chest and abdomen on peak pressure tolerated. They demonstrate that chest and abdominal binding for prevention of pulmonary over-expansion does allow greater pressures to be sustained. It demonstrates that pulmonary pathology such as adhesions can affect the ability to tolerate high intrapulmonary pressures. It also provides gross evidence of the movement of air from the alveolus into interstitial tissue planes and further into the mediastinum. There is evidence here that this process begins at around 60-100 mmHg in both bound and unbound cadavers. The authors conclude that overexpansion of the lungs is the cause of pulmonary barotrauma. They set the peak static pressure limit in unsupported humans as 80 mmHg, and in supported at about 190 mmHg. This study has several limitations. The five subjects of the experiment are

not representative of the population of interest, the active military aviator, and in general are advanced in age. The study cannot account for the differences between living and dead tissues. Finally, these limits are for static pressures and not dynamic pressures. In general though, these are reasonable numbers guiding an investigator to approach pressures above these with caution and adequate safety measures.

Decompression studies are reported in the diving medical literature. In comparison to studies done at altitude, these pressure differentials occur over long periods of time, 1-2 minutes. The pressure range is much greater, many atmospheres, at high final pressures, versus fractions of atmospheres at low initial and low final pressures.[18] It is similar in the manner that the entire body is exposed to the pressure change and the damage is caused by the delay in equilibration of the gas in various portions of the body--with only the gasses in the thorax producing fatality.[19] These studies are not helpful in solving the problems encountered in flight above 50,000 feet, for the reasons stated above, and are not reported here.

It is clear now that our currently understood limits of pulmonary overpressure are relevant to a more easily explored environment of slow decompressions in a diving environment, or aviation environment decompressions of a mild nature. While these limits are useful guidelines, they do not completely or adequately predict the performance of man at altitude in a rapid or explosive decompression environment.[28]

Methods

All research was conducted through the Strughold Library USAF SAM, Brooks AFB, TX. Initial searches were conducted through MEDLINE and Defense Technical Information Center (DTIC). Articles were selected for inclusion in the review based upon at least one of the following criteria: presentation of original research with data from human or animal models directly related to pulmonary overpressure; review article with summary of data or research questions answered to date; bibliography; presentation of human respiratory performance limit, or original model or hypothesis concerning pulmonary overpressure. These guidelines were meant to be very broad initially, to encompass all of the possible articles on the subject. A secondary search was conducted beginning with selected textbook bibliographies from: Aviation Medicine, Second Edition, John Ernsting and Peter King[66]; Fundamentals of Aerospace Medicine, Roy DeHart.[50] In addition, bibliographies from AGARD publications in Aerospace medicine, No. 312,[2] 322[4] and 516[3], and the Bibliography on Aviation Medicine, Volume I[6] were also used as a starting point for the secondary search. References cited in the bibliographies relating to any of the four criteria listed above were obtained. These bibliographies were also searched. As long as the bibliography search produced new articles, the search was continued. Finally, the decompression sickness data base at USAFSAM/High Altitude Protection Laboratory was manually reviewed.

Data analysis was conducted by identification of the basic elements needed to determine the nature of the decompression. Information required to adequately describe the decompression is: the initial and final atmospheric pressure, and the time allotted for the pressure change. All times were given in units of seconds. In cases where time ranges were given, the time for the pressure change was assigned the longest duration possible in the experimental conditions. Pressures were reported in pounds per square inch (psi), millimeters of mercury (mmHg), and altitude equivalents(feet above sea level). All pressures were converted to mmHg using the United States Standard Atmospheric Pressure Table 1.[10] Where pressure ranges were given, the pressure ranges giving the smaller pressure changes were used. Where data could be summarized without loss of important detail, it was. All references used, and complete discussion of all data points used can be found in the technical report being published through Armstrong Laboratory. This report summarizes and extrapolates beyond the report findings.

Results

The maximum pressure that can be safely tolerated by the human pulmonary system in a dynamic overpressure situation is unknown. This measurement has not been performed. Evidence suggests that the unsupported chest wall of the human population can safely support 80 mmHg static and dynamic overpressure of the lungs. This is not to be misunderstood as an absolute limit for the population. It is likely that the pilot

population of the United States armed forces could tolerate higher dynamic pressures, due to a lower prevalence of pulmonary pathology than in the general population.[5,71] Safe static pressures in the human population wearing chest and abdomen support devices is at least 190 mmHg. It is probable that pilots wearing well designed life-support equipment could endure higher pressures without permanent injury.

The range of decompressions tolerated by human subjects and documented in the literature reviewed is presented in Graph 1, Human Decompressions, Pressure Range vs Time. Each bar represents at minimum one safe decompression. These represent the decompressions most commonly employed in experimental protocols. They are also the most extreme decompressions available in the pressure range, change and time indicated. Decompressions longer than 5 seconds in duration were not included as these were not in the realm of explosive decompressions.
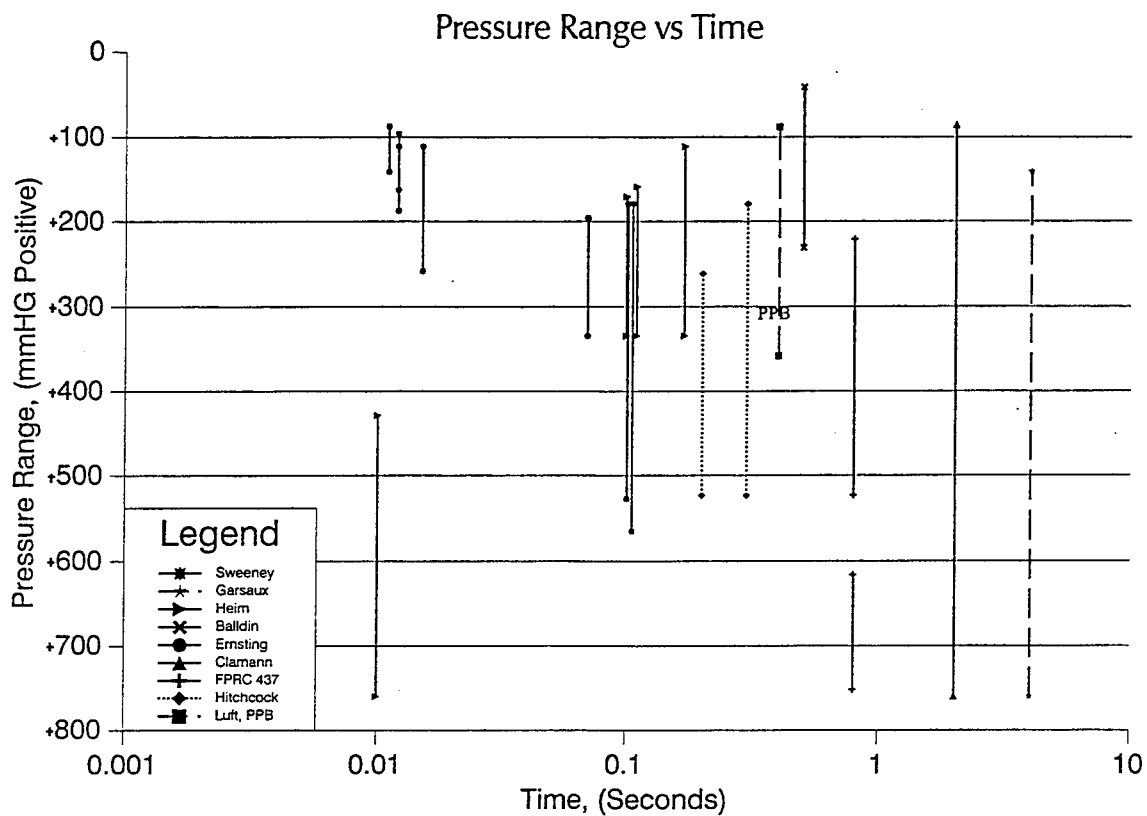
Graph 2, Human Decompressions, Pressure change vs Time, presents the same data seen in Graph 1, but uses only the absolute pressure difference on the y axis. This does not account for differences in the decompression due to density effects and effects at altitude. The heavy line drawn on the graph represents the limit of human experience. From this graph, it can be concluded that a healthy aviator without additional life-support can tolerate the 5 psi cockpit differential pressure change safely as long as it is 0.05 seconds in duration or greater. There are no experimental decompressions less than 0.01 seconds in duration. Safe decompressions can be bounded by pressure changes less than 100 mmHg in 0.01 seconds, 375 mmHg in 0.1 seconds, and 675 mmHg in 2 seconds. The single decompression at 330 mmHg and 0.01 seconds was reported by J.W. Heim in 1939 in an introduction to his paper.[49] The subject tolerated the decompression without complaint. This is the lone human data point suggesting that healthy human subjects more than currently thought. Beyond these points, published data was not identified.

Results of animal experiments are graphed on Graphs 3-4, Animal Decompressions. Limits of human decompression experience have been superimposed for comparative purposes. The most significant data is summarized on the graphs; duplicate data points were excluded for clarity. The data indicates decompressions without serious temporary or permanent effects from as brief as 0.01 sec to 2 sec over a wide range of pressure changes. Both graphs demonstrate limits of safe animal decompressions. In general, decompressions less than 0.01 second are not well explored, and appear to be unsafe over large pressure changes. Changes of pressure of one atmosphere in less than one second can cause death, but may be tolerated in a large percentage of the population. Changes of one atmosphere in greater than one second are generally tolerated by animals and are not presented except in graph 1. These graphs generally show that human data is less extreme than the animal data.

There are limits to the animal data that could be used to guide future human experimental work. Hall, 1957, definitively demonstrated that there are definable limits to tolerable dynamic overpressure exposures.[47] These experiments are connected point to point to form the "Limit of Animal Safe Decompressions." Pulmonary toxicity occurs below this line, as this approximates a lethal exposure to 50% of the animals exposed. Lethal exposure provides a more readily determined endpoint of toxicity in animals than does quantification of pulmonary hemorrhage. This is why this line can be drawn as a relatively firm limit of exposure.

The validity of animal models for estimating tolerance limits for human pulmonary toxicity is likely good. In the only study that evaluates the static and static-supported pressure limits of a large variety of animals, J.P. Henry, in 1945 determined the "strength of the alveolar wall was of the same order as that of the capillary bed, namely 50-100 mmHg."[51] The animals varied in size from mice to steer. The primary difference was in the rigidity of the thorax. Dogs and steer performed most like the cadavers in the Malhoutra study.[60] The flexibility of the chest wall in cats, rats and mice limited their applicability to the human situation. These smaller animals will tend to underestimate the human tolerance for a given static pressure situation. Use of well-designed flight gear, providing chest and abdominal counterpressure, increases human pulmonary overpressure tolerance primarily through increased thorax rigidity. Use of well-designed flight gear should increase the safety margin for tolerance of a 7 psi decompression in 0.01 seconds in duration and greater.

Graph 1: Human Decompressions
Pressure Range vs Time

Graph 2: Human Decompressions.
Pressure Change vs Time
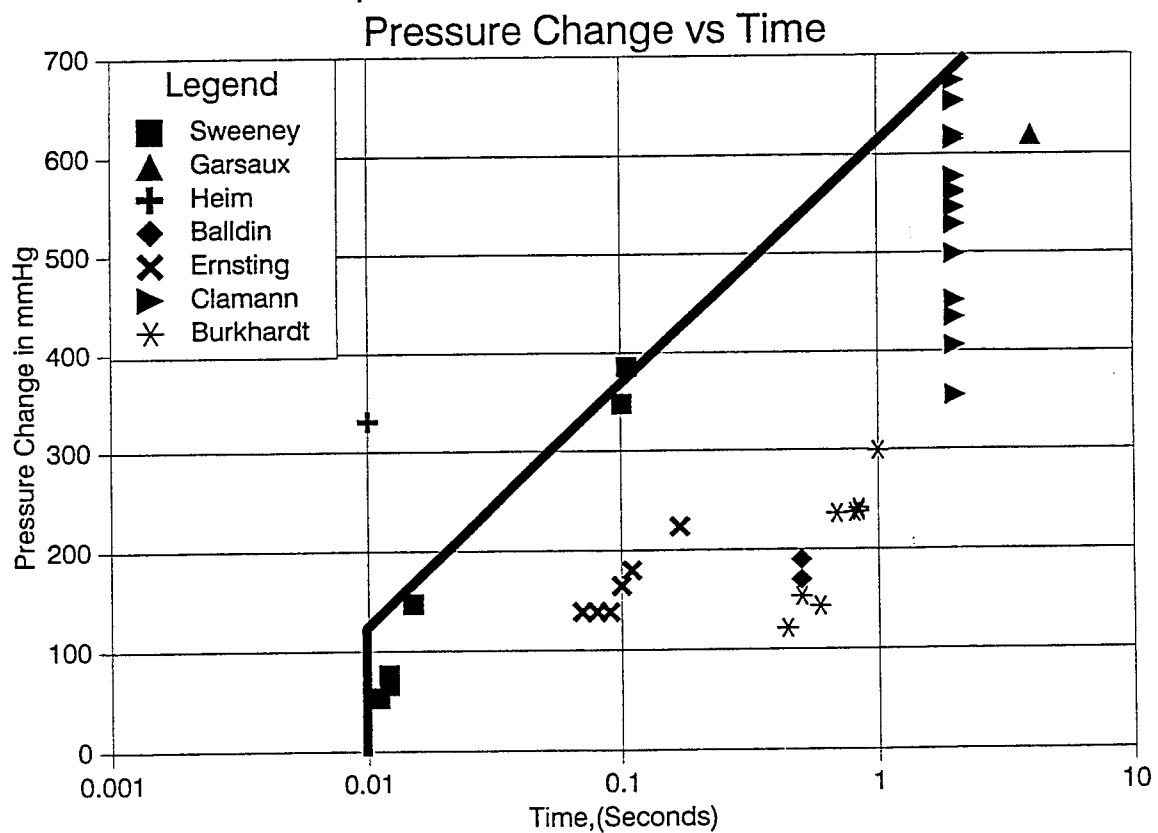
Graph 3: Animal Decompressions
Pressure Change vs Time

Primary Author

✕	Bancroft
◼	Burkhardt
◆	Edelmann, 1946
▶	Gelfan
▲	Edelmann, 1952
✚	Fegler
●—	Hall
●	Hall(Safe)
★	Rosenbaum
✶	Vail

Graph 4: Animal Decompressions.
Pressure Range vs Time

Legend

◼ Bancroft	✚ Edelmann, 46	✕ Edelmann, 52	◆ Fegler
▲ Gelfan	★ Hall	● Hall & Corey	✕ Rosenbaum
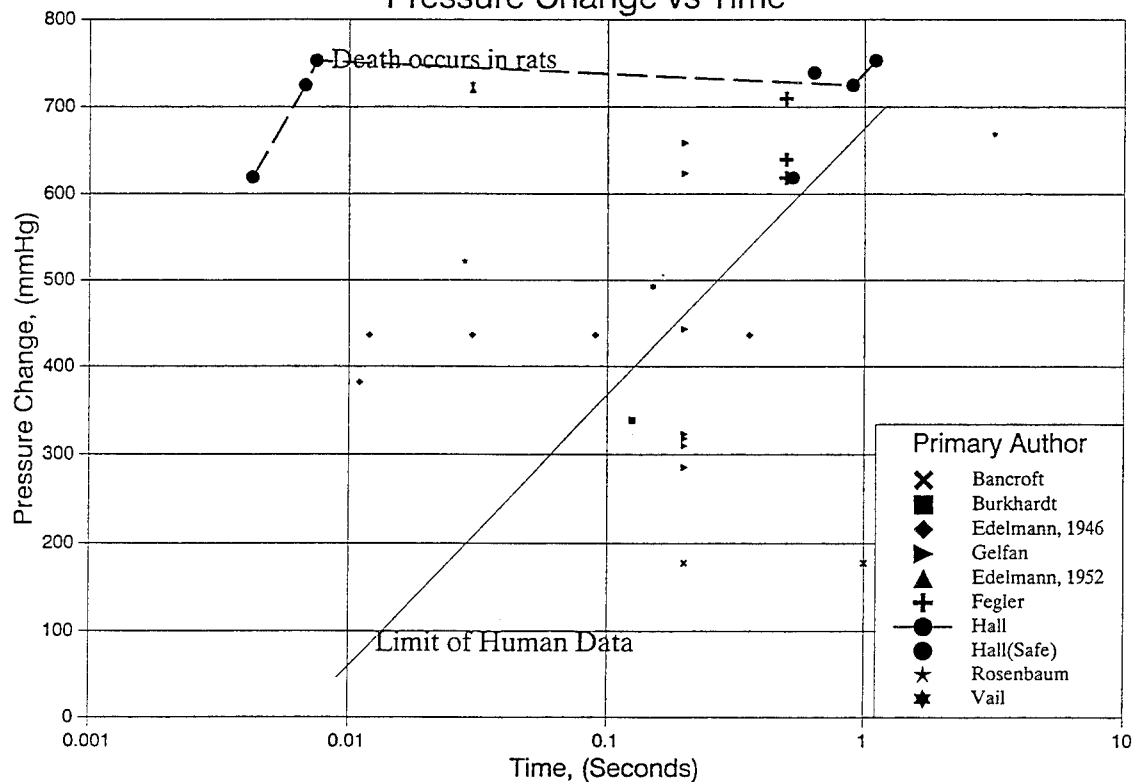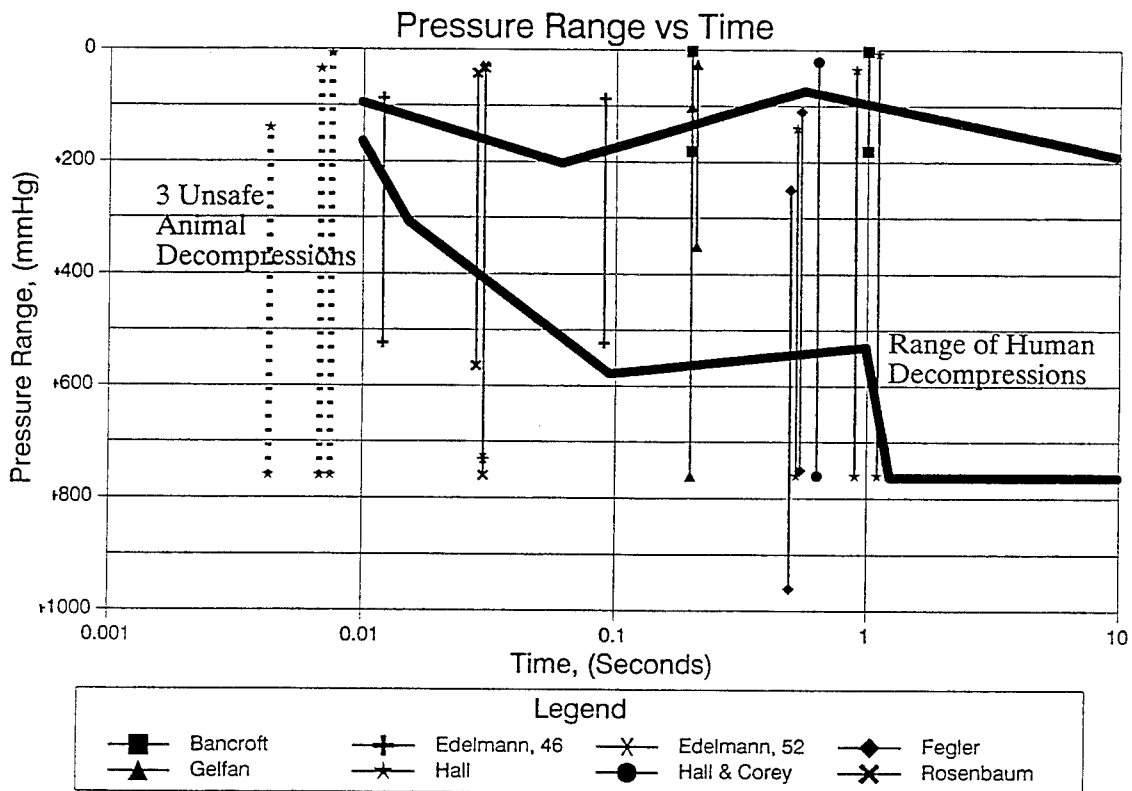
In summary, the safe limits for static pulmonary pressure are conservatively set at 60-100 mmHg, and for supported static pressures at 170-190 mmHg. These limits do not account for dynamic overpressure situations. By current human data, the unsupported aviator could tolerate the standard 5 psi, (260 mmHg), overpressure occurring in 0.06 seconds or greater, a 6 psi, (314 mmHg), overpressure in 0.08 seconds or greater, and a 7 psi, (361 mmHg), overpressure in 0.1 seconds or greater. Animal evidence suggests that greater pressures could be tolerated in humans with minimal toxicity. Using the J.W. Heim data point and the animal data, the active military aviator should be able to tolerate about a 6.5 psi, (330 mmHg) overpressure in durations greater than 0.01 seconds. Pilots will be exposed to pressures beyond the currently accepted safety limits, and most will survive. It will be the responsibility of flight surgeons and physiologists to document and publish these events in order to adjust our current understanding to improve the capability of future aircraft.

Research Problems

Investigation of the isolated effects of pulmonary overpressure is confounded by the effects of hypoxia,[15,37,48] ebullism[32] and evolved gas phenomena--decompressions sickness.[20,30] It is complicated by the variability of the biological system under study,[39,65] including alterations from normal induced by disease processes.[31,42] Poor study design has also plagued research in this area. They can affect future studies if steps are not taken during study design to eliminate or control for their effects.

Conclusions

Alveolar oxygen content may be maintained through increase in pressure, increase in oxygen content or both.[12] High-altitude flight in advanced fighter aircraft without pressure suits will require both increases in oxygen content and pressure. At 37,500 ft, 100% oxygen without additional pressure will maintain an oxygen pressure of 159 mmHg.[53,73] Above this point, additional pressurization will be required in the form of either cabin pressurization, or directly applied positive breathing gas pressure, or both.[34] Both methods have implications for pulmonary overpressure.

The physiologist and engineer control barometric pressure change around several parameters.[33,53] Decompression sickness (DCS) is a primary factor determining maximum cabin altitude. In general, cabin altitude should not exceed 22-24000 feet (321-294 mmHg) for flights of short duration to prevent DCS.[38] The other factor contributing to the decision of maximum allowable cabin altitude is explosive decompression. The greater the differential between the cabin altitude and the ambient aircraft altitude, the greater the potential for injury to the pilot from decompression.[33] All decompressions to altitudes in excess of 40,000 ft (141 mmHg) may be considered as decompressions terminating at this pressure altitude. The prevention of hypoxia in the steady state at these heights demands that the absolute pressure within the respiratory tract not fall below the equivalent of 40,000 ft, 141 mmHg.[53,73] What must be considered in decompressions to altitudes above 40,000 feet (141 mmHg) is the peak pressure experienced by the pulmonary system, and the life-support equipment designed to ensure adequate oxygen pressure and chest counterpressure.

In addition to use of pressure to maintain adequate alveolar oxygen at altitude, positive pressure breathing for G protection is now being employed in our combat forces.[43,44,48,59,61,68] While pressures are theoretically limited to safe static pressures, the possibility of exposure to overpressures exist. These may occur through dynamic overshoots of the system, or through cabin pressurization failure during high-G maneuver. Knowledge of a safe dynamic overpressure would aid designers in providing a maximally effective system for lowest cost.

It should be noted that all of the data presented indicate completed decompressions without permanent injury, except where noted. The range of decompressions is from 0.01 seconds to over 5 seconds, with pressure changes of only 100 mmHg to almost one full atmosphere. In general, humans were not exposed to conditions beyond what might be expected in operational conditions.[56] This points the way for future research requirements.

To date, the exact limits for safety for explosive decompression have not been defined. The boundaries of the human experience are evident on all graphs.

In summary, the paper presented here define the current limits for human pulmonary overpressure. Animal models were used to validate theory and physiologic pulmonary events of decompressions. The animal data provides evidence for a decompression limit; evidence that extreme conditions may be survivable; evidence that damage attributed to decompression at altitude is often due to anoxia, and suggests repeated decompressions are more hazardous than single decompressions. In addition, animal data confirms flight equipment can affect the outcome of a decompression. Controlled, experimental data on human tolerance to dynamic pulmonary overpressure is limited to a few studies. These studies do not suggest that the human tolerance for dynamic pressures is significantly different from the animal models used. Carefully constructed studies will be required to define the limit of pulmonary toxicity in the dynamic overpressure scenario. Determination of this limit is vital to maximize our performance capabilities in operational aircraft.

Recommendations

In my opinion, the following should be done now:

1. Future aircraft designs intended for frequent and extended flight above 50,000 feet should be designed with a cabin differential pressure of up to 7 psi. The chosen altitude should be the minimum pressurization needed to effectively eliminate the risk of decompression for the flight profiles projected for the aircraft. A cabin pressurization of around 6 psi should meet this objective for most aircraft.
2. Life-support equipment should be included to provide additional protection against the explosive decompression. This can be done by a pressure valve which will keep the chest and abdomen counterpressure within 15 mmHg of the mask pressure during an explosive decompression.
3. Acquisition policy should be changed sufficiently to consult the life-support and human physiology divisions prior to setting aircraft life-support specifications.

Future research objectives should include the following:

1. Defining the biologic variability surrounding pulmonary pressure limits.
2. Defining the incidence and type of pulmonary and pleural pathology in the aircrew population. The goal is defining appropriate selection and retention standards for aircrew in these aircraft.
3. Defining the performance of current life-support equipment in the explosive decompression environment at high altitude.
4. Creating a model to adequately predict the outcome of decompressions in currently unexplored environments.

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Discussion

MAJ KREBS: In summary, I don't think that concern over pulmonary overpressurization should limit our discussion regarding changing the cabin pressurization schedule. I think the differential could safely be raised to 7 PSI. If it is decided that is too extreme, in order to get the benefit of the added pressure with a reduced decompression sickness incidence, you might consider raising it to 6.2 psi.

DR. SEARS: Just a comment. In 1953, I was an outside observer/lock operator on a rapid decompression training chamber flight at Randolph AFB that exposed the trainees (young medical officers) over a 2-3 second decompression from 8,000 to 30,000 feet. A fatal accident occurred with death attributed to closure of the airways during the decompression. It was never determined whether he was in the act of swallowing, coughing or intentionally held his breath against the rapid decompression. Death occurred within three minutes from a massive pneumothorax and air embolism. The differential pressure was 6.5 PSI; shortly thereafter the differential was reduced in training to approximately 4.5 PSI, which is equivalent to the standard training profile from 8,000 to 22,000 feet. Indeed, problems can occur if the guy has a well-fitted high-pressure mask on without a pressure vest and can't get rid of the gas in the lung. Or if he's swallowing, clearing his ears, or anytime he has his glottis closed.

MAJ. KREBS: What I failed to state here, that is noted in the Technical Report, is that there are several criteria that require consideration. The numbers given assume at the start of decompression the lung is at functional residual capacity and the glottis is open, i.e., there are no other obstructions that would prevent the loss of gas from the lung. The reality is that if you meet the right criteria, you can kill a person with less than what we're currently using.

DR. ACKLES: Do we have any estimates regarding the time of decompression if an aircraft loses its canopy? Has anyone ever recorded a canopy loss in flight? It seems to be critical.

MAJ. KREBS: Dr. Ernsting earlier presented Dr. Violette's modeling criteria where he used a time constant which relates the volume of the cabin to the opening. I assumed the absolute worst case scenario going through a 5 PSI pressure differential instantaneously.

PROF. ERNSTING: The French, in the late 1950s, had a whole series of ruptured canopies in flight where they actually recorded the pressure changes. Dr. Ackles, I don't know whether you are familiar with that paper. I can't recall the details, but they published it in the French aviation medicine journal.

MAJ. KREBS: That paper is not in the Technical Report. I wasn't able to find it.

PROF. ERNSTING: I've got a copy of it at home, but I can't recall the time of decompression.

DR. ACKLES: Okay, so it has been measured.

PROF. ERNSTING: You can also estimate from canopy jettison testing the rate at which the hole appears. We will ask British Aerospace to give us some data to see if we can determine the rate of canopy separation as part of the ejection sequence. On the Harrier aircraft, the whole canopy is explosively removed so you could expect a 0.05 second decompression.

DR. BOMAR: Some time back, I calculated the volume of a fighter canopy, mathematically compressed it into a sphere and allowed it to re-expand. My recollection is it's 70 microseconds for the center of that sphere to reach the ambient pressure. The front is sonic and you can calculate the time it takes.

MAJ. KREBS: I only used data that people have actually tolerated, hoping to come to something useful.

Alternative Methods for Pressure Breathing

R E. Moon, M.D.
B.W. Stolp, M.D., Ph.D.

Introduction

Positive pressure breathing (PPB) can increase altitude tolerance by producing an increase in inspired, and hence alveolar, PO_2. The magnitude of this increase is most closely related to mean airway pressure. This airway pressure elevation has effects which can reduce tissue oxygen delivery and result in significant pilot morbidity:

Reduced Cardiac Output

High levels of PPB can result in hypotension, and reduction in cardiac output (see Physiology of Pressure Breathing by B.W. Stolp). The mechanism of this cardiopulmonary impairment is not totally understood but could include: (1) reduced venous return due to elevated intrathoracic pressure; (2) a increase in pulmonary vascular resistance leading to increased right ventricular afterload; (3) shift in interventricular septum due to right ventricular volume overload, resulting in impaired left ventricular filling. All of these mechanisms have been implicated in the hypotension associated with positive pressure ventilation, and especially the application of positive end expiratory pressure (PEEP), in acutely ill patients.

Hyperventilation

In clinical medicine mild hyperventilation occurs as a result of application of PPB (usually termed continuous positive airway pressure or CPAP) (Wherry, et al, 1980). With the introduction of PPB into military use in the 1940s a much greater increase in ventilation was observed, occasionally leading to tetany and loss of consciousness, even at a pressure of only 19 mmHg (Hall, 1953). It appears to be totally involuntary and largely uncontrollable even in well trained individuals. At low levels of PPB the mild hyperventilation observed is probably compensatory for an increase in physiological dead space (Folkow & Pappenheimer, 1953). However, at high levels (30 - 60 mmHg) there is only a small increase in respiratory dead space and the observed hyperventilation cannot be explained on this basis. At high PPB levels there can be significant hypocapnea, which because of cerebral vasoconstriction may further reduce tissue oxygen delivery. As a result there may be impaired pilot performance and loss of consciousness.

Pulmonary Barotrauma

Pulmonary barotrauma (PBT) can also occur, which can be manifested as interstitial emphysema, pneumomediastinum, pneumothorax or even arterial gas embolism. Effective thoracic counterpressure should theoretically minimize this complication, provided the device prevents the expected increase in lung volume. Nevertheless, a pressure gradient probably still exists at the interface between the pleural cavity and the mediastinum, since straining maneuvers, for example in pregnant women during labor or saxophone playing, can cause pneumomediastinum even in normal individuals (Macklin & Macklin, 1944; Karson, et al, 1984; Snyder, et al, 1990). Major PBT could also occur in pilots if the counterpressure mechanism failed.

In fact, the human body is a poor transducer of absolute pressure; pressure *gradients* and tissue distention are therefore the most likely explanations for the phenomena observed and also the likely cause of major complications like PBT. One can conclude that placement of the pilot in a full-pressure garment could provide the pilot with the necessary augmentation of alveolar PO_2 without adverse cardiorespiratory effects. Enclosure of the entire body within a pressurized suit would be equivalent to providing a form-fitting, pressurized cockpit. However because of practical limitations to pilot dexterity this solution may be unacceptable.

The challenge is to design a PPB system which provides an adequate increase in alveolar PO_2 while minimizing the detrimental effects. Previous studies and operational experience are summarized below.

(1) Pressurized suit would have the effect of merely increasing cockpit pressure. The more closely a partial coverage suit mimics a full-pressure suit, the less physiological derangement will occur.

(2) Diminished cardiac output and inability to sustain arterial pressure has been shown in part to be alleviated by inflation of the leg bladders to a pressure three to four times the breathing pressure. However, the increased intrapulmonary blood may augment the risk of pulmonary barotrauma.

(3) Periodic reductions from a mask pressure of 60 mmHg can augment blood pressure. Phasic swings in mask pressure also seem to augment venous return and enable subjects to maintain mean arterial pressures at sustained PPB levels to 60 mm Hg, especially under simulated altitude conditions (low breathing gas density/high compressibility).

(4) At high levels of PPB (^{3} 30 mmHg) marked hyperventilation occurs predominantly due to some other effect than PPB-induced increased dead space.

(5) The reduction in arterial PCO_2 resulting from hyperventilation reduces cognitive and psychomotor performance, possibly in part mediated by a reduced O_2 delivery to the brain.

(6) The cardiovascular system and other soft tissues do not transduce absolute pressure, but rather stretch, which results from pressure *differentials*.

(7) Ventilation/perfusion abnormalities induced by positive pressure breathing will induce a significant discrepancy between end-tidal and arterial gas tensions.

(8) Intrapulmonary shunt caused by G-induced atelectasis may worsen gas exchange and lower the altitude at which sudden decompression may cause critical hypoxia.

Alternative Methods

Counterpressure limited to specific areas of the body (e.g. COMBAT-EDGE, TLSS) using a flexible garment can provide an acceptable "hyperbaric environment", though with some uncovered areas allowing for development of pressure gradients, for example the arms, perineum, neck and the skull foramina.

In addition to augmenting the area of coverage of the protective suit, improvement in tolerance to PPB may be engineered using the following possible approaches, singly or in combination:

(1) Application of PPB in a manner which results in greater cardiovascular tolerance.

(2) Decreasing the reflexive hyperventilation.

Since the augmentation of alveolar PO_2 is dependent upon mean alveolar pressure, it is unlikely that any change resulting in a reduction in mean mask pressure will provide adequate support of arterial oxygenation. However, if the change results in less reduction in organ blood flow, overall tissue oxygen delivery could be maintained. One method would be to change the pressure application from continuous to phasic. This could be done in two ways. Pressure could be increased during inspiration (analogous to pressure support ventilation) or expiration (analogous to PEEP or expiratory positive airway pressure, EPAP). Conventional continuous PPB is illustrated in Figure 1. Mask pressure is maintained close to Po at all times, with minor fluctuations in pressure

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Fig. 1: Continuous Positive Airway Pressure
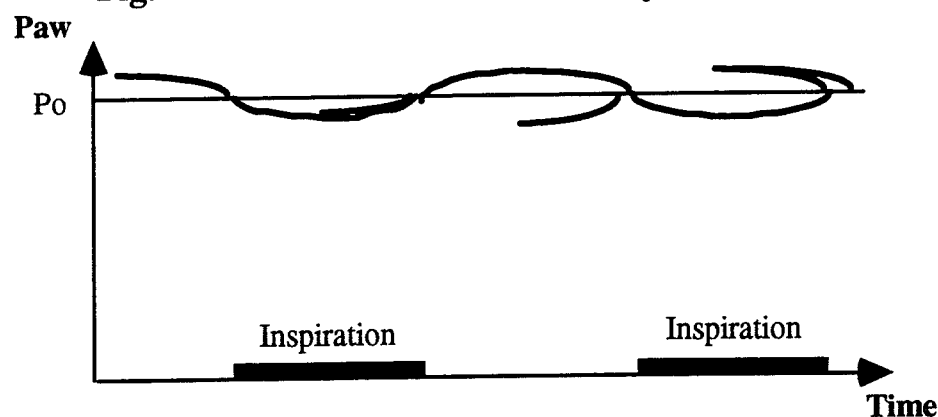
due to the breathing resistance of the apparatus. Figure 2 shows inspiratory pressure support. When the device detects the onset of inspiratory flow, mask pressure is increased from P1 to P2, providing some inspiratory assist. The phasic changes in mask pressure, and hence pleural/media-stinal pressure, might augment venous return, and hence cardiac output. A form of this mechanism was previously implemented as the "Pneumolator" (Barach, 1947). One possible disadvantage of this implementation is that it may cause greater involuntary hyperventilation than continuous PPB. However, a systematic comparison of this method with continuous PPB using modern control systems is warranted.

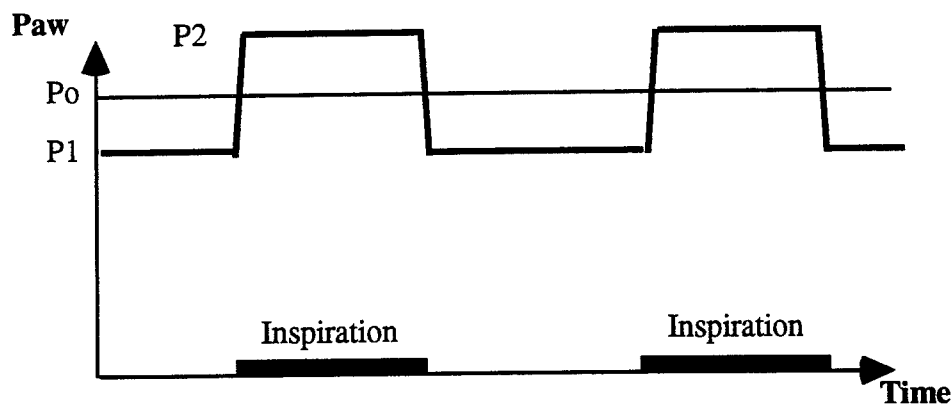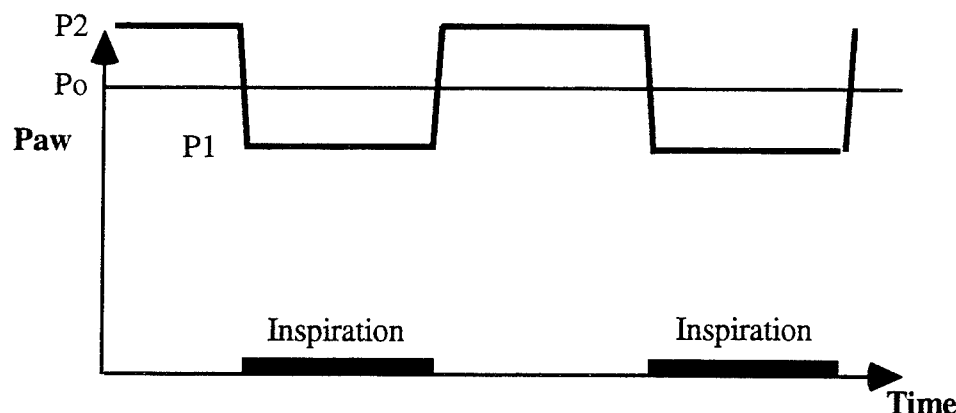## Fig. 2: Inspiratory Pressure Support

Figure 3 shows the converse of inspiratory pressure support, phasic expiratory pressure increase. In this implementation the pilot would inhale from P1 then exhale against an elevated pressure P2.

Fig. 3: Expiratory Phase Increase

Studies in normal volunteers (Schlobohm, et al, 1981) have shown that although EPAP of 10 cmH_2O results in greater work of breathing than CPAP of the same magnitude, it is accompanied by greater intra-esophageal pressure fluctuations. Gherini (Gherini, et al, 1979) demonstrated that while CPAP 15-20 cmH_2O resulted in involuntary hyperventilation but PEEP did not. Therefore, in pilots, expiratory phase pressure increase may result in higher cardiac output than continuous PPB, perhaps without the troublesome hyperventilation.

High-Frequency Oscillation

Pulsing the respiratory system at high frequency may be additionally useful in two ways. First, oscillating the airways of normal volunteers with volumes of 30-40 ml at 15 Hz resulted in significant prolongation of mean breath hold times, from 56 to 264 seconds (Butler, et al, 1980). This suggests that oscillating the airway at subsonic frequencies may possibly attenuate PPB-induced hyperventilation. Second, oscillation timed to the electrocardiogram may offer additional benefit. Using anesthetized dogs Matuschak (Matuschak, et al, 1988) demonstrated that during hypovolemia stroke volume could be increased by approximately 10% when airway pulses were applied during late diastole. In patients with congestive cardiomyopathy, when compared to traditional mechanical ventilation or random application of airway pulses, synchronization of airway pulses with the upstroke of the radial artery pulse augmented cardiac output by 30% (Pinsky, et al, 1987). Superimposition of high-frequency pressure impulses upon the breathing circuit in a PPB system is conceptually simple and this principle could easily be tested in a breathing system designed for pilots.

Summary

Replacement of continuous PPB with a system providing phasic pressure alterations has the potential to augment cardiac output and tissue oxygen delivery, while possibly minimizing hyperventilation. Systematic laboratory studies of these alternative techniques are required.

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Discussion

MAJ. DIESEL: Did you normally see a change in the respiratory pattern?

DR. MOON: Yes, when CO_2 was added the ventilation went up.

MAJ. KREBS: You plotted the phasic pressures in a sinusoidal manner rather than using peak pressures at three times average pressure. I thought the peak pressure required would be too high.

DR. MOON: Well, I think the peak pressure, or shall we say the amplitude of the pressure phases could be adjustable to whatever one wants. The additional beneficial effect of oxygenation is a function of mean airway pressure. So I don't think we can get away with lowering the average pressure. But you could use whatever phasic swing you desire. It may only be a matter of 5 millimeters of mercury. I just don't know; we'd have to do the experiments.

DR. GOODMAN: It's an interesting concept, oscillating, pulsing the PPB. Do you think the primary effect is in the lung, allowing the pulmonary vasculature to refill and supplying volume to the left ventricle?

DR. MOON: It could operate at the level of either the right or left ventricle. The traditional explanation has been that it applies a boost to the left ventricle, but of course that's not necessarily the case. The reduction in pressure during the negative part of the phase may be augmenting right ventricular filling, or the increase could augment right ventricular emptying as well. So I'm not sure exactly where it's acting but presumably it's on ventricular performance.

DR. GOODMAN: I think you made a very important point that a lot of what we're seeing could really be on the right side of the heart. I haven't been able to look at the right side of the heart since our monitoring technique focuses on the left ventricle. As further support to your findings, in some of the nuclear studies, we were able to look at right ventricular shapes and the data suggested a septal shifting. There has been some pulsating suit studies at the Canadian Space Agency, and some modeling in the G suit area. The concept there is to pulsate the G suit to allow more efficient venous emptying. There are some people who believe that if you constantly pressurize a bladder over veins you may form a constriction which prevents blood from leaving the vein. So the pulsating effect might have a role in G suit function as well.

DR. MOON: I agree. In fact, one could actually pulsate the counterpressure vest if this thing seems to work at a pulmonary level. If it does indeed work, then it might be easier to pulsate the vest rather than trying to pulsate the airway.

PROF. ERNSTING: Aviation physiological literature is full of the effects of adding CO_2 during hypoxia to increase performance. We're all very aware that many of the symptoms and performance decrements you get in moderate hypoxia can be corrected by adding CO_2 to the inspired gas. I'm sure you are aware of Dr. Chris Lambertsen's studies measuring PO_2s and saturation's at 42,000 feet in which he added various levels of CO_2.

As an aside, I'm no expert on the cytochrome oxidase technique you mention, but we've not been very impressed with its use in the chamber or centrifuge. Dr. Glaister brought the system back after his tour here at the Armstrong Laboratory and he has not had much success using it to look at cytochrome oxidase levels. I don't know what the Brooks AFB experience has been.

Pressure Breathing Inflation Schedules/Ratios

DP Gradwell, Sqn Leader RAF, Ph.D., MB, ChB

Introduction

The technique of raising the pressure at which an individual breathed was first adopted clinically in 1878 in an effort to relieve asthma and pulmonary oedema. During WWII, however, Gagge and his colleagues investigated its novel use as a means of preventing severe hypoxia at high altitude.(1) They experimented with a system in which an individual breathed 100% oxygen at an increased pressure of between 15 and 25 mmHg. It was then possible for the subject to tolerate exposure to an altitude of 50,000 ft for a few minutes.

Early observations on the respiratory and circulatory effects of pressure breathing included comment on the reversal of the normal respiratory cycle with full distension of the lungs produced by a positive pressure of 20 mmHg. PPB also induces a wide range of effects on soft tissues in the head and neck. Radiographic studies of the chest whilst pressure breathing have shown both elevation and expansion of the thoracic cage, descent of the diaphragm and a reduction in pulmonary vascular markings, suggesting a reduction in pulmonary blood volume. Spirometric studies of the lung have revealed that pressure breathing gives rise to an increase in total lung capacity, and particularly to increases in expiratory reserve and residual volumes, with a proportionate reduction of inspiratory reserve volume.(2)

Intra-pleural pressure rises as intra-pulmonary pressure is increased by pressure breathing and the rise in arterial blood pressure associated with pressure breathing has been attributed to the direct effect of increased intra-pleural pressure on the left ventricle. The relationship between the rise in arterial BP and applied breathing pressure is not a simple one: in general, the rise in BP has been seen to be as great as the rise in breathing pressure perhaps because of elastic recoil in the lungs and the influence of extra-thoracic baroreceptors trying to lower blood pressure as a response to increased distension.

Thoracic venous pressure also rises with pressure breathing and then greatly exceeds the venous pressure in the peripheral veins leading to a sharp reduction in venous return. The increased transmural pressure in limb blood vessels will inevitably lead to a greater movement of fluid from the vascular compartment into the tissues, and to a net loss in circulating blood volume.

The degree of reduction in circulating blood volume is directly related to the breathing pressure and the duration for which it is applied.

Pressure breathing has been found to give rise to a tachycardia related to the magnitude of the applied breathing pressure. A fall in effective circulating blood volume, an impairment of venous return to the chest, and radiographic evidence of a reduced blood volume in the heart and pulmonary vessels, with inadequate filling of the right atrium, as a result of pressure breathing, could all be expected to contribute to the ensuing tachycardia.

Sustained pressure breathing will induce syncope. The prevention of pressure breathing syncope would appear to depend upon the maintenance of an appropriately elevated arterial blood pressure, and upon limiting the degree of induced tachycardia.

The Development of Counterpressure Assemblies

Bazett and MacDougall, in 1942, had remarked upon the potential benefits of trunk counterpressure in improving tolerance to pressure breathing.(3) Counterpressure was applied with a close fitting, inelastic, waistcoat containing an inflatable circumferential bladder. The bladder was inflated with breathing gas through a T-piece in

the mask hose and, by this means, the pressure across the chest wall was balanced. This assembly was used to raise the operational ceiling of high-flying aircraft by 4,000 ft. Barach et al commented on the possibility of adding elasticated lower limb coverage to an enlarged counterpressure jacket to make a complete counterpressure assembly.(4) Such assemblies were noted not only to make breathing easier, but also to prevent expansion of the abdominal wall and pooling of blood in the splanchnic circulation.

Counterpressure has been shown to have a beneficial effect on the adverse cardiovascular consequences of pressure breathing, but the form of the counterpressure has a significant influence over its efficacy. Ernsting showed that torso counterpressure reduced the incidence of pressure breathing syncope, and made of pressure breathing more tolerable.(2) An assembly consisting of jerkin and anti-G trousers has become the standard form of partial-pressure assembly for protection up to 56,000 ft in use in the RAF over many years in a number of aircraft types. It has also been the model for similar assemblies used by other nations and remains in use in the UK and with NASA.

Roxburgh made brief reference in 1956 to an apparent benefit of raising anti-G trouser inflation to a pressure greater than breathing pressure and a "barometric anti-G valve" was adopted for use in some RAF high-altitude interceptor aircraft in the 1960s.(5) On exposure to high altitude, this device produced inflation of the anti-G trousers to approximately 40 mmHg greater than breathing pressure. Larsson and Stromblad sought to develop a partial-pressure assembly in which less body surface area was covered by inflatable bladder than had previously been accepted and inflated the anti-G trousers to a pressure higher than that of the breathing gas.(6) Balldin showed that rapid decompressions from 29,500 ft to 65,617 ft could be accomplished using a two-pressure suit.(7)

At the same time Ackles and his colleagues conducted an extensive ground level study into the degree of protection offered by three different pressure breathing assemblies: the standard RAF mask, jerkin and anti-G trousers system, a Canadian pressure waistcoat and anti-G trousers, (both assemblies had a uniform inflation pressure, equal to breathing pressure, in the upper and lower garments), and, finally, the Swedish system with anti-G trouser inflation pressure of 3.2 times breathing pressure.(9) The study was later extended to examine the physiological effects of applying differential counterpressure with the Canadian and British assemblies, again with anti-G trousers pressures of 3.2 times breathing pressure. Results from this series of experiments suggested that a greatly improved level of physiological protection, as evidenced by the elevation and maintenance of arterial blood pressure and the minimisation of pressure breathing tachycardia, was conferred by the application of increased counterpressure in the anti-G trousers. The level of protection offered by the two-pressure Swedish assembly was ranked highly against that provided by the standard British assembly. Furthermore, when the two-pressure inflation technique was used in conjunction with the British system a still greater degree of protection was achieved. The Canadian group concluded there was benefit to be gained by inflating anti-G trousers to four times breathing pressure.

Altitude Protection Requirements of Future Fighter Aircraft

We have examined the improvement in physiological protection by applying a greater level of counterpressure in the lower garment. We assumed the current standard RAF assembly provided the minimum acceptable level of protection and was a suitable control in our studies. The experimental counterpressure assemblies comprised a waistcoat containing a circumferential chest bladder and anti-G trousers but with the inflation pressure of the two garments controlled in a pre-determined ratio relationship with respect to the breathing pressure. The influence of the pressure difference between upper and lower garments was examined in three ratios from 1:1 to 1:4 (upper garment inflation pressure always equaling breathing pressure), so encompassing the range suggested by other groups The protection conferred by standard (control) and experimental assemblies was compared and ranked.

We adopted the premise that the maintenance of blood pressure, and indeed its elevation during pressure breathing, along with the ability to minimise any increase in heart rate as indicative of the adequacy of the cardiovascular protection provided by partial-pressure assemblies. The maintenance of cardiac output is impaired by inadequate venous return during pressure breathing. Therefore an ideal partial-pressure assembly would support the circulation by maintaining venous return and reducing cardiovascular work. A ratio for blood pressure

responses to pressure breathing was derived to aid the comparison between various assemblies and counterpressure relationships.

Experimental Methods

Initial studies were conducted to examine the influence of counterpressure assemblies on lung volumes and capacities. Pressure breathing in the absence of counterpressure is known to cause an increase in total lung capacity, a small increase in tidal volume but a large increase in expiratory reserve volume, at the expense of the inspiratory reserve volume. The standard RAF counterpressure assembly consisting of a jerkin covering the whole of the trunk and combined with conventional anti-G trousers was able to minimise these changes in lung volumes during pressure breathing. Other assemblies consisting of counterpressure vests, covering only part of the chest, waistcoats applying counterpressure to the front of the chest only or circumferential waistcoat and anti-G trouser counterpressure at the breathing pressure. All were less effective in preventing the changes in lung volumes seen during pressure breathing than that achieved by the jerkin assembly. But a combination of a circumferential waistcoat and anti-G trousers inflated to a pressure three times breathing pressure was found to be at least as effective in preventing the lung volume changes of pressure breathing as was the jerkin assembly. (Figure 1) On the basis of these results it was considered acceptable to proceed with an examination of the circulatory effects of pressure breathing and of high-altitude exposure using the experimental waistcoat/anti-G trousers combination.(10)
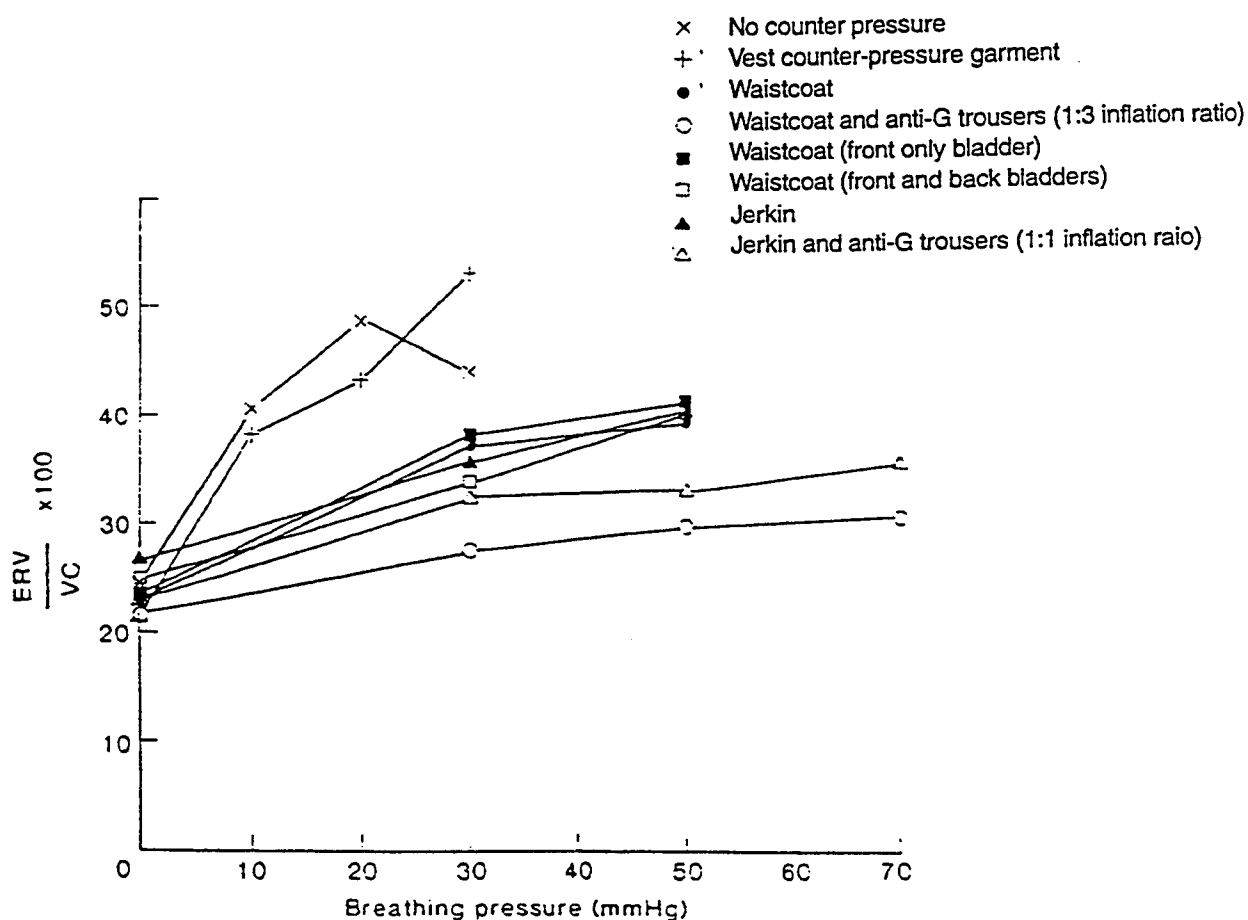

Figure 1. Effect of counterpressure coverage on the relationship between expiratory reserve volume (ERV) and vital capacity (VC) during pressure breathing.

Measurements for studies into pressure breathing at high altitude were made as follows:

Electrocardiograph (ECG) and Heart Rate (beats·minute^{-1})
Blood pressure (BP),
Arterial Oxyhaemoglobin saturation percentage (SaO2%)
Respiratory gas analysis: partial pressures of oxygen, nitrogen & carbon dioxide
                             in the mask cavity (PO2, PN2, & PCO2)
Inspiratory flow (L·min^{-1})
Inspiratory ventilation (L·min^{-1}, from integration of flow)
Chamber altitude, (feet)
Mask Cavity Pressure (mmHg)
Anti-G trouser pressure (mmHg)

Blood pressure measurements were made by the non-invasive Finapres technique, having previously confirmed the acceptability of this method under pressure breathing conditions in comparison with intra-arterial blood pressure recording.(8)

Six healthy male subjects (aged 31 - 40 yrs), able to tolerate pressure breathing at up to 70 mmHg above ambient for periods of at least two minutes, completed this series of studies. At each of the four altitudes to which the subjects were decompressed each wore either the standard Royal Air Force Mk4 Partial Pressure Jerkin and anti-G trousers, or the same type of anti-G trousers in combination with an experimental chest counterpressure waistcoat. In the latter case, however, subjects were decompressed with ratios between breathing pressure and anti-G trousers inflation pressure of 1:1, 1:3 or 1:4.

On completion of pre-oxygenation subjects were rapidly decompressed in the hypobaric chamber to one of the final altitudes. The subject remained at that altitude for a period not exceeding two minutes prior to recompression to ground level. The degree of pressure breathing to which the subjects were exposed was related to the final altitude as follows:

Final altitude	Mask Cavity Pressure
45,000 ft	30 mmHg
50,000 ft	45 mmHg
55,000 ft	60 mmHg
60,000 ft	70 mmHg

Ground-Level Pressure Breathing Experiments

An extensive programme of low-altitude or "ground-level" studies was also carried out. For these experiments the subject was seated in the chamber capsule but breathing gas being delivered from a supply hose was connected, via a Douglas tap, to the outside of the chamber. The subject was instrumented in a similar manner as for high-altitude experiments. The hypobaric chamber was then slowly decompressed to establish a pressure differential across the chamber equivalent to the desired level of pressure breathing. Opening the Douglas tap would then provide the correct degree of pressure breathing to the subject, and the wide bore connection through the chamber wall provided a very low-resistance system. The whole series of pressure breathing exercises required at high altitude were duplicated in these ground-level studies. Additionally pressure breathing at 70 mmHg mask cavity pressure wearing the experimental counterpressure assembly with a garment inflation ratio of 1:1 was carried out in ground-level studies but for reasons of safety not included in the high-altitude programme.

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Results

Effects of Pressure Breathing and Enhanced Counterpressure on Blood Pressure

One hundred and thirty-five high-altitude rapid decompressions (RDs) were successfully accomplished in the course of this study with only two failures to complete two minutes of pressure breathing at the final altitude. A further 250 ground-level PPB experiments were conducted using the "through the wall" technique.

Rapid decompression and initiation of PPB were associated with immediate changes in the arterial pressure wave-form and the recorded blood pressures (BP). With the onset of PPB arterial pressure was elevated and its waveform modified, the dicrotic notch became more prominent and, with a change in heart rate, the systolic peak became more sharply defined. Pulse pressure was initially maintained then diminished during the next respiratory cycle. Thereafter an exaggerated variation in pulse pressure with respiration was observed. Furthermore, although mean BP fell from the peak attained immediately after the RD it quickly returned to elevated levels. In many experiments BP showed a continued small upward progression during the remainder of the PPB exposure.

Pressure breathing experiments conducted at ground level with the same four positive breathing pressures, resulted in responses very similar to those observed in high-altitude experiments.

The high-altitude and ground-level PPB experiments, in the standard partial-pressure assembly, comprised two series from which it was possible to observe the BP response to pressure breathing across a range of breathing pressures. The results showed a progressive increase, above resting levels, of systolic, diastolic and mean blood pressures with increasing levels of PPB. When comparing the effects of using the experimental counterpressure assembly with various lower garment inflation ratios no significant differences were found between the BP responses shown when wearing the standard assembly and the experimental one with an inflation ratio of 1:1. A marked difference between the influence of standard and experimental assemblies was seen, however, in the significant ($p<0.001$) reductions in pulse pressure observed during pressure breathing when using the latter assembly at a ratio of 1:1. When higher pressures were used in the lower garment the pattern of changes in blood pressure, pulse pressure and heart rate differed from those seen with uniform (1:1) upper and lower garment inflation pressures. Rises in blood pressure with pressure breathing were much more marked. The increased effect of enhanced lower garment inflation pressure was found to persist with the use of an inflation ratio of 1:4, although the level of significance was, perhaps surprisingly, lower in a number of cases ($p<0.05$ or <0.01). The use of this ratio did not result in any further increase in the degree of changes induced, but rather that the blood pressures observed were less elevated than those manifested with an inflation ratio of 1:3.

The comparative effects of the standard and experimental assemblies, at all three ratios, on the responses of mean blood pressure to all four breathing pressures examined are shown in Figures 2 (from high-altitude PPB results) and 3 (from ground-level PPB results). The positive change in mean BP increased as the breathing pressure rose in control experiments with the standard assembly. Use of the experimental assembly at an inflation ratio of 1:1 was associated with elevations of mean BP smaller than, but not significantly different from, control studies. Increasing the ratio to 1:3, however, brought about a substantial increase in the response shown. No further significant increase occurred if the inflation ratio was raised to 1:4. These relationships were assessed by a multiple analysis of variance which revealed that the pattern of responses shown, and the significant positive relationship ($p<0.001$) between breathing pressure and increases in BP, was consistently maintained by the experimental assembly at all four breathing pressures examined. Although the mean arterial pressure changes are plotted in Figures 2 and 3 the same consistency in results was found in both systolic and diastolic pressures.

Figure 2. Histogram of comparative changes in mean arterial pressure during pressure breathing at altitude in standard and experimental (Exp) assemblies. Garment inflation ratios are stated in relation to their use in experimental assemblies. Note: The experimental assembly was not used at 60,000 ft (70mmHg breathing pressure) with a 1:1 ratio.
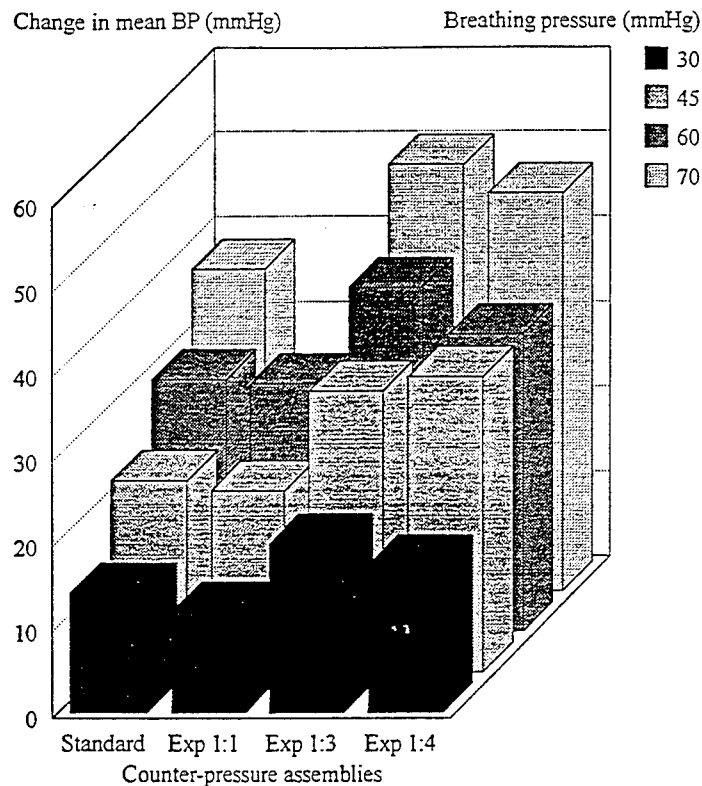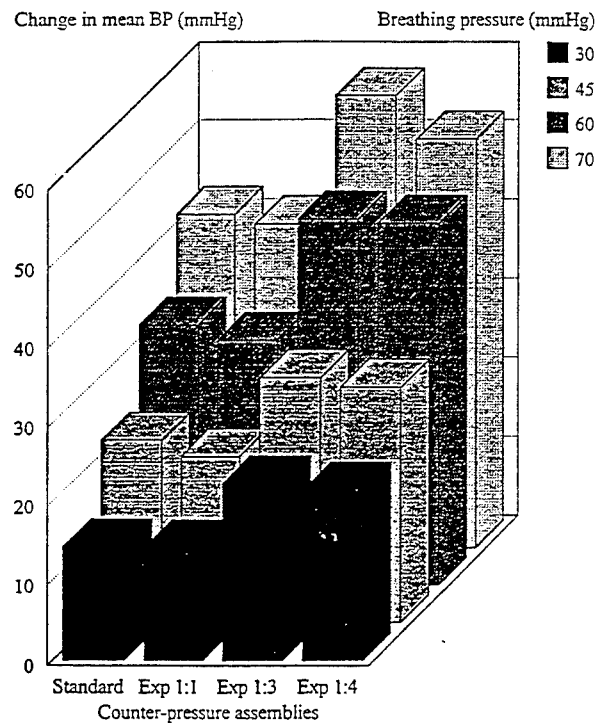

Figure 3. Histogram of comparative changes in mean arterial pressure during pressure breathing at altitude in standard and experimental (Exp) assemblies. Garment inflation ratios are stated in relation to their use in experimental assemblies. Note: The experimental assembly was not used at 60,000 ft (70mmHg breathing pressure) with a 1:1 ratio.

Effects of Pressure Breathing and Enhanced Counterpressure on Heart Rate

The onset of pressure breathing caused an immediate increase in heart rate. Furthermore, in the control studies performed with the standard partial-pressure assembly the heart rate showed a continued overall rise throughout the period of pressure breathing. When the experimental assembly was used with an inflation ratio of 1:1, at high altitude or ground level, the heart rate increases were commonly greater than those observed with the standard assembly. With enhancement of lower garment inflation pressure, however, the steady and progressive increase in heart rate with rising breathing pressure was modified, rising by a much smaller degree at each level of PPB, both at altitude and in ground-level studies. It is evident that the progressive increase in heart rate with breathing pressure is modified by the use of both inflation ratios of 1:3 and 1:4. An analysis of variance showed not only that the change in heart rate with breathing pressure was significant ($p < 0.001$), but also that both the higher inflation ratios had a significant effect in reducing the tachycardia associated with pressure breathing in comparison with both the standard assembly and with the experimental assembly at an inflation ratio of 1:1. (Figure 4).

Standard assembly
Experimental assembly (ratio 1:3)
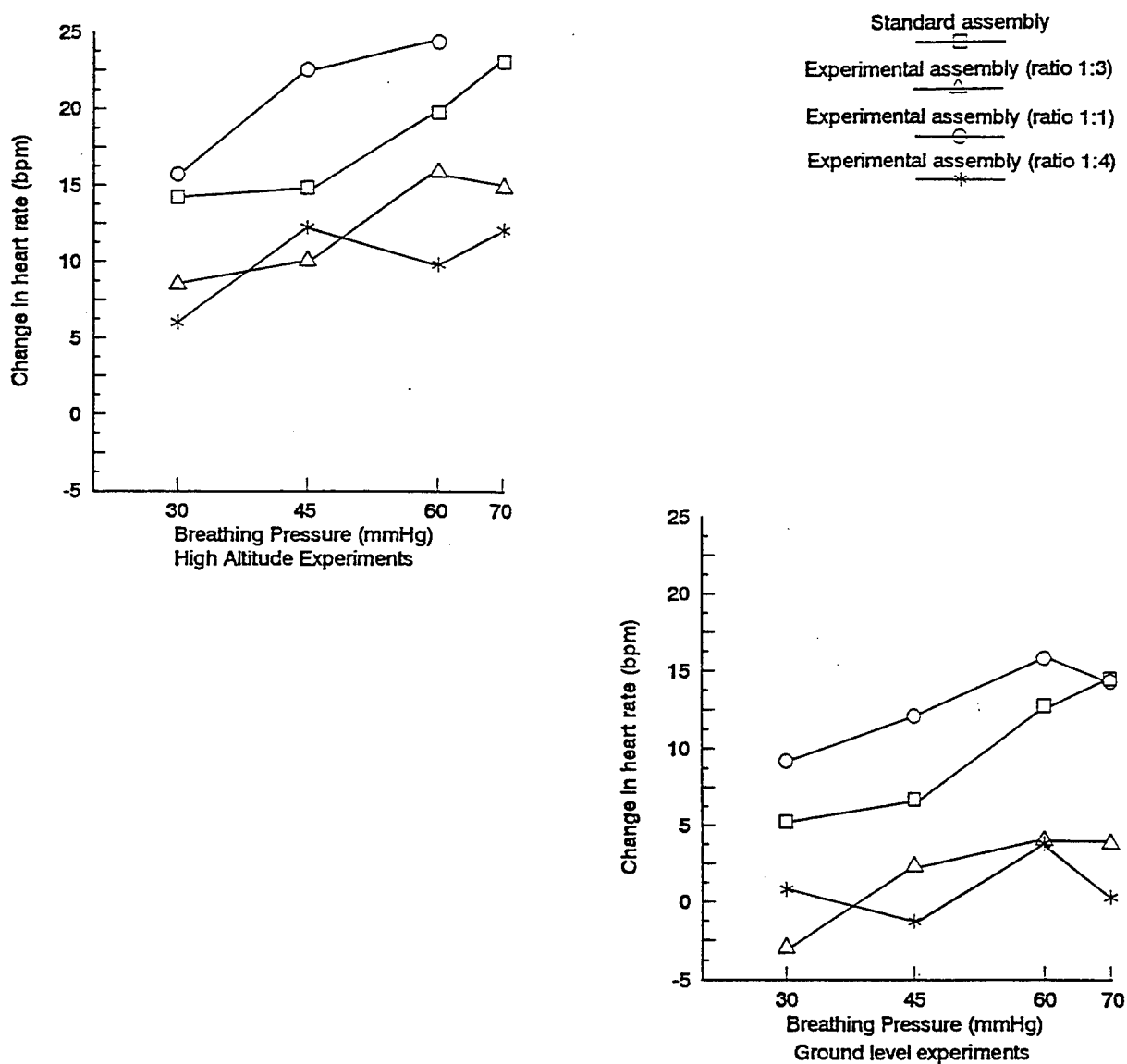Experimental assembly (ratio 1:1)
Experimental assembly (ratio 1:4)

Figure 4. Changes of heart rate during pressure breathing and the influence of counterpressure inflation ratios in ground-level and high-altitude studies.

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Effects of Pressure Breathing and Enhanced Counterpressure on Respiration

Examination of the respired gas and flow records confirmed that most subjects were able to slow their respiratory frequency from resting levels during even the most stressful high-altitude experiments. Notwithstanding the reduction in respiratory frequency from a mean of 11.8 breaths·minute^{-1} to 9.5 breaths·minute^{-1}, in experiments to a final altitude of 60,000 ft inspiratory ventilation was elevated significantly (p<0.001) and associated with a fall in the alveolar partial pressure of carbon dioxide. The mean ventilation at the base altitude (22,500 ft) of 13.0L(BTPS)·min^{-1} increased to 27.3L(BTPS)·min^{-1} whilst pressure breathing at 70 mmHg mask cavity pressure in subjects wearing the standard assembly. The use of the experimental counterpressure assembly at the same altitudes had no significant effect on the ventilation recorded.

Applying a correction for water vapour pressure to recorded values reveals a mean PAO2 during pressure breathing at 70 mmHg mask cavity pressure at 60,000 ft of 53.6 mmHg for subjects wearing the standard assembly, with a corresponding PACO2 value of 25.3. These compare with the base altitude values for PAO2 of 229.5 mmHg and PACO2 of 36.9 mmHg. When the experimental assembly was worn the alveolar partial pressures were not significantly different from those noted for the standard assembly and mean PAO2 values of 51.8 mmHg and 50.7 mmHg were found with the use of the experimental assembly at inflation ratios of 1:3 and 1:4 respectively. As would be expected, base altitude PAO2 levels were not significantly different from the control.

The degree of respiratory comfort associated with the use of the control and experimental counterpressure assemblies was not formally assessed, but subjects reported breathing to be easier when the lower garment was inflated to three times mask cavity pressure than when using the former assembly in control experiments. The improvement in the tolerability of pressure breathing with increased lower garment counterpressure, however, was not sustained at an inflation ratio of 1:4. At that ratio the abdominal bladder was considered to be intrusive, making respiratory movement more difficult and aggravating any discomfort from gastrointestinal gas expansion associated with rapid decompression.

Full-Coverage Lower Counterpressure Garments

Conventional anti-G trousers cover approximately 30% of the lower body surface below the umbilicus. In contrast, full-coverage anti-G trousers (FCAGT) cover approximately 95% of the lower body. In a series of ground-level experiments the degree to which these garments improve circulatory tolerance to PPB was studied. The arterial blood pressure and heart rate responses to two minutes positive pressure breathing was examined at five levels of FCAGT counterpressure at various breathing pressures. The results suggest that the minimum acceptable level of FCAGT pressure required to provide optimal circulatory support during positive pressure breathing is 1.5 times the breathing pressure. Increases in the level of FCAGT inflation pressure above 1.5 times the breathing pressure offered no additional circulatory support to pressure breathing. A FCAGT inflation pressure greater than 1:3 may compromise circulatory support.

The use of FCAGTs resulted in a more marked blood pressure response than had been observed with conventional anti-G trousers, at each inflation pressure examined. Whereas use of a 1:1 ratio with conventional anti-G trousers had resulted in a smaller increases in systolic BP than those seen in diastolic pressure, this relationship was reversed when full-coverage anti-G trousers were substituted at the same ratio. Thus, since the rise in systolic pressure exceeded that seen in diastolic pressure the changes observed emulated those found when enhanced lower garment inflation pressures were used with conventional anti-G trousers. Furthermore, the use of FCAGTs had a noticeable effect on the changes in pulse pressure, reversing the previously observed reduction in pulse pressure during PPB.

Changes in heart rate with pressure breathing were also influenced by the use of FCAGTs. Even with an inflation ratio of 1:1 heart rate rises were very much smaller than when conventional trousers were used and even fell when higher inflation pressures were used.

Discussion

Comparisons of the degree by which blood pressure responses approach the summation of resting BP and the applied breathing pressure are simplified by the development of a single expression for such a characteristic. This can be calculated for any breathing pressure and any degree of change in blood pressure (systolic, diastolic or mean) and expressed as a ratio of dBP/PPB. Thus a rise in mean BP of 30 mmHg when pressure breathing at 30 mmHg could be expressed as a ratio dBP/PPB of 1.0. Conversely, a ratio of less than 1.0 would indicate blood pressure had risen less than breathing pressure whilst pressure breathing, and paradoxically could be considered to reflect relative hypotension.

Calculation of the average (for the six subjects) mean arterial dBP/PPB ratios in high-altitude and ground-level control experiments using the standard jerkin/anti-G trousers assembly gives values of 0.50 and 0.54 respectively. Thus there was a marked disparity between the increases in intra-pleural pressure and elevations of arterial pressure. Furthermore, although the extent to which BP rose in response to a specific level of PPB in these control experiments was of a broadly similar order in systolic, diastolic and mean pressures, the dBP/PPB ratios for increases in diastolic pressures consistently exceeded the corresponding systolic dBP/PPB ratios at all breathing pressures examined.

In considering the mechanisms brought into action by pressure breathing two of the most important are the arterial baroreceptors and the cardiopulmonary pressure receptors. They provide an integrated system for the detection of arterial, and cardiac filling pressures. The carotid and aortic baroreceptors are stretch receptors, their activity stimulated by increases in transmural pressure as arterial pressure rises. However, if a baroreceptor region is unable to distend no increase in its activity will be recorded. During pressure breathing intra-thoracic pressure is elevated at least as much as the BP, so aortic baroreceptors will not be exposed to an increase in transmural pressure and would therefore be unresponsive or, if the target BP is not achieved, may even be expected to respond to relative hypotension and any fall in pulse pressure. In contrast, the carotid baroreceptors, outside the chest and unsupported by counterpressure, will be subjected to an elevated BP and stimulated by increased transmural pressure.

A reduction in the total area of counterpressure coverage, such as the substitution of a pressure waistcoat for a jerkin, has been associated with disadvantages in respect of protection against lung distension as measured by spirometry, although increasing the lower garment inflation pressure may improve the degree of protection. Comparison of cardiovascular responses to pressure breathing whilst wearing standard and experimental assemblies with an upper to lower garment inflation ratio of 1:1, at altitude and in ground-level studies, showed broadly similar responses with few statistically significant differences between the BP responses observed. However, the use of a waistcoat and anti-G trousers at the same uniform inflation pressures showed a not statistically significant tendency for smaller rises in arterial pressure and lower pulse pressure during PPB. Furthermore, this result, taken in conjunction with the co-incident greater degree of tachycardia during pressure breathing, pointed to the suggestion that the reduction in area over which counterpressure was applied resulted in a greater circulatory disturbance, less efficient and effective counterpressure and a further reduction in venous return. Subjective accounts of the competence of the counterpressure assemblies, given by those wearing them during pressure breathing, concurred with these observations. These results were in keeping with the hypothesis that any reduction in the counterpressure applied would also reduce the degree of protection conferred on the circulation. The mean dBP/PPB response ratios, calculated in the manner described, show that the standard assembly performed better in the control studies than the experimental assembly at an inflation ratio of 1:1.

In contrast, the efficiency with which the experimental assembly with enhanced lower garment inflation pressures improved circulatory support was considerable. As reported in the previous chapter, BP and heart rate responses to PPB were greatly influenced by the use of inflation pressure ratios of 1:3 and 1:4 between upper and lower garments. Under these circumstances diastolic blood pressure rose considerably more during PPB than observed when using the standard counterpressure assembly, but the increases in systolic pressures were even

greater, generally exceeding the changes in diastolic pressure. In addition, the pulse pressure was noted to be maintained or even increased by the adoption of enhanced lower garment counterpressure. That these results reflected a profound change in the degree of protection provided to the circulation was also supported by the marked contrast observed in the heart rate responses to pressure breathing. The increases in heart rate in relation to a specific level of PPB were approximately 50% of those seen using the standard assembly. The mechanisms responsible for the responses to pressure breathing must therefore be modified by an increase in lower body compression.

With enhanced lower garment counterpressure a greater pressure was exerted on the capacitance vessels and the large muscle beds of the lower limbs. This applied an effect comparable to venoconstriction and an enhanced skeletal muscle pump, and redistributed a greater venous volume centrally. Enhanced venous return increases cardiac pre-load, improves atrial and ventricular filling and hence, by Starling's law, improves stroke volume and maintains cardiac output. Thus, although the presence of positive pressure in the chest is an impediment to inward venous flow, enhanced lower body counterpressure alleviates the reduction in venous return from the lower limbs. Furthermore, heart rate increased less during PPB with enhanced lower garment inflation pressures. This may suggest that enhanced lower body counterpressure is more able to correct an imbalance caused by high-positive intra-thoracic pressures impeding venous return, rather than distend the atrial receptors. Pulse pressure was shown to increase generally during PPB with enhanced garment inflation ratios, an effect of increased venous return and an enlarged central blood volume.

The largest lobe of the anti-G trousers' bladder is the abdominal one, and this also inflated as counterpressure was applied at the onset of pressure breathing. PPB itself causes an increase in intra-abdominal pressure, even in the absence of any form of counterpressure, but the application of abdominal counterpressure substantially greater than breathing pressure will increase intra-abdominal pressure still further. This might be expected to encourage venous return and displace blood towards the chest, but the relationship between intra-abdominal pressure and venous return is complex and subject to modification by respiratory movement. Nonetheless, the high intra-abdominal pressure generated will favour improved venous return provided lower limb counterpressure is at least as great as abdominal.

Increasing the lower garment inflation pressure to three times breathing pressure had effects suggestive of significantly improved protection of the circulation. Increasing that ratio to four times breathing pressure in conventional anti-G trousers did not produce still greater levels of circulatory support and with FCAGT no significant improvement was noted using an inflation ratio of 1:3 rather than 1:2 or 1:2.5.12 The explanation for the further effect of increased garment coverage in FCAGTs may be related to both the greater surface area over which counterpressure was applied and the circumferential nature of the bladder. The effect on the venous compartment was enhanced, reducing the potential for venous blood to be held in uncompressed regions of the lower limbs and abdomen, therefore increasing the pre-load still further and resulting in a more substantial increase in systolic pressure. The increase in pulse pressure with PPB, rather than its fall, and the change in the heart rate response also suggest a greater efficacy with which venous return is sustained.

Compression of the arterial tree and constriction of inflow with FCAGTs was only increased to a modest degree in comparison with conventional anti-G trousers at the same inflation pressure, and therefore the increases in after-load were similar. This may account for the disparity in the increases in systolic and diastolic pressures associated with the replacement of conventional anti-G trousers by full-coverage ones.

Increases in heart rate when pressure breathing, wearing the experimental assembly incorporating FCAGTs, were much smaller and in some cases heart rate even fell during pressure breathing. This too may be considered to reflect the more efficient support of venous return, acting through cardiopulmonary receptors, and given the approximation of the observed blood pressure to target values, any aortic baroreceptor driven increase in heart rate would be reduced.

Conclusions

The standard counterpressure assembly of partial-pressure jerkin and anti-G trousers inflated to breathing pressure, as used by pilots of high-altitude aircraft in the UK and elsewhere, can make tolerable for limited periods the use of breathing pressures of up to 70 mmHg, at altitudes up to 56,000 ft. An experimental counterpressure assembly consisting of waistcoat and anti-G trousers, when inflated uniformly to breathing pressure, that is with an inflation ratio of 1:1, provided no greater support to the cardiovascular system than the standard assembly.

The adoption of differential counterpressure, however, with the lower garment inflated to a pressure several times greater than the mask pressure, rendered pressure breathing at high altitude more tolerable, and was associated with no episodes of pre-syncope. Moreover, inflation of the lower counterpressure garment to three times breathing pressure caused highly significant improvements in the physiological protection conferred by the assembly. This was observed as an arterial pressure elevated closer to the sum of resting BP and applied breathing pressure, the more satisfactory maintenance of pulse pressure and the minimising of the tachycardia associated with pressure breathing. Increasing the lower garment inflation to four times breathing pressure did not further improve the protection provided. In subsequent studies of the influence of using full-coverage anti-G trousers (FCAGTs) it was observed that an inflation ratio less than 1:3 was able to effect similar improvements in circulatory tolerance to pressure breathing.

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Discussion

SQN LDR GRADWELL: As an additional comment, we use the term, "Jerkin", when we apply counterpressure to the whole trunk. The term, "Waistcoat", is used when chest only is pressurized and the term, "Vest", is used when only very minimal coverage to the chest is applied.

DR. SEARS: Does your work with the uniform pressure garment ratios suggest that G suit ratios might be reduced?

SQN LDR GRADWELL: I can't make that suggestion. Maybe others can.

COL. HILL: Yes, there was a short study, with very few subjects, where the suit pressure was backed off to about 8 PSI at +9 Gz. I think the break point was somewhere around 8.5 or so.

DR. SEARS: So maybe it can be reduced?

COL. HILL: Yes, but not a lot.

PROF. ERNSTING: You've seen Wg. Cmdr. Prior's study which was fully reported in the Pensacola AGARD Meeting where he tried all sorts of G pressures, G suit pressures and breathing pressures and found similar results. Both laboratories agree on what the ratios ought to be during PPB.

COL. HILL: Dr. Prior's study was more robust in fact and conclusive.

Physiological Responses to Long Duration Positive Pressure Breathing and Explosive Decompression Up to 72,000 Ft.

W. D. Fraser, M. Sc.

Introduction

The use of positive pressure breathing with 100% oxygen to protect against hypoxia following exposure to altitudes above 40,000 ft. has been under active research since World War II (Bazett & MacDougall, 1942; Ernsting, 1965; Holness et al. 1980; Buick et al. ,1991; Fraser et al., 1994).

Though the pulmonary consequences of PPB are well documented, including increased respiratory frequency, increases in minute volume, and fatigue of the expiratory muscles (Ernsting, 1965), the use of chest counterpressure can, to a large extent, ameliorate these problems. However, there is still the adverse effect of PPB on the cardiovascular system, characterized in part by progressive circulatory collapse leading to syncope, as a result of the pooling of blood in the lower extremities, a decrease in venous return, a decrease in cardiac output, and a fall in systemic blood pressure (Ackles, et al. 1978). The cardiovascular consequences of PPB have been regarded as the limiting factor in allowing for extended use of very high levels of PPB (Ernsting, 1965), with the physical discomfort of the procedure also a major factor in discouraging subjects. Though the use of chest counterpressure and the anti-G suit reduces these adverse consequences, syncope will occur after still relatively short periods of time with very high levels of PPB.

Background

Ackles et al. 1978 have shown that a critical factor in preventing the development of severe cardiovascular dysfunction is the counterpressure in the anti-G suit. Using a pressure in a low-coverage anti-G garment such as the CSU 15, which is 4 times the PPB pressure, results in a dramatic suppression of the adverse effects of PPB on cardiac function, compared to an anti-G suit/PPB ratio of 1:1.

Though extensive work has been undertaken in the development of advanced life-support systems such as TLSS, ATAGS, and EAGLE, which use extended coverage anti-G suits and PPB to enhance acceleration protection, little work had been undertaken to examine the ability of these new designs to provide enhanced protection against the adverse consequences of PPB, or examine their effectiveness in providing get-me-down protection following explosive decompression at high altitude.

Current Status

Since the advanced anti-G systems might be required to provide protection against severe hypoxia, as well as acceleration, we commenced a series of what were regarded as relatively routine experiments to (i) document the effects of extended coverage anti-G garments on the cardiovascular consequences of PPB and (ii) ensure that such anti-G suit/PPB jerkin systems would provide similar protection against hypoxia, as that provided with the limited coverage anti-G suit and CF jerkin/PPB ensemble (Ackles, this workshop).

In our first series of studies (Goodman et al. 1992) we examined the cardiovascular consequences of PPB in a group of 12 subjects while they wore either the Combined Advanced Technology Enhanced Design G Ensemble (Combat Edge or CE) system or the older experimental Tactical Life-Support System (TLSS) which has approximately 45% greater bladder coverage in the anti-G suit. This was a straightforward experiment to establish

the protective limits of the garments against the consequences of PPB. Though we anticipated some improved cardiovascular protection with the TLSS garment, we chose an upper limit for PPB exposure of 10 minutes, confident that the subjects or the medical monitoring officer would abort the experimental runs due to the severe discomfort of the procedure or severe decrements in blood pressure, long before they had reached that limit.

To our surprise, all 12 subjects were able to withstand 10 minutes of 60, 70, and 88 mmHg PPB while wearing the TLSS ensemble, with 5 of the 12 subjects able to complete the 10 minutes of 88 mmHg PPB while wearing the CE ensemble, and 9 of the subjects were able to complete the 60 mmHg PPB exposure. As well, subjects were able to tolerate the discomfort due to the PPB, and perform the Mannikin psychomotor task throughout the exposures. Exposure to PPB for that length of time is certainly not a pleasurable experience, but can be tolerated in *well trained subjects.*

In our second series of studies (Fraser *et al.,* 1993), we examined the cardiovascular consequences of PPB in a group of 6 subjects while they wore either the Tactical Life-Support System (TLSS), or the US Navy Enhanced Anti-G Lower Ensemble (EAGLE) and USAF Advanced Tactical Anti-G System (ATAGS). This study was done in order to complete our database on extended anti-G suit designs. This time, we anticipated that all three garments would allow PPB exposures of somewhere over 10 minutes, and chose 20 minutes as the maximum duration, not expecting any subjects to reach that new limit.

However, all six subjects completed 20 minutes of PPB with the ATAGS system at 60 and 70 mmHg PPB sessions and four subjects completed the 80 mmHg PPB exposure. Five of the subjects completed the 60 mmHg PPB session and four of the subjects completed the 70 and 80 mmHg PPB session with the EAGLE garment. Five of subjects completed the 70 mmHg PPB exposure with the TLSS garment. In all but one case the runs were aborted by the medical officer due a drop in the mean arterial pressure. In all but one of these cases the subjects felt they could have continued. In one case the subject aborted a TLSS run due to pain in the urinary bladder. One subject was responsible for five of the eight aborts. This same subject had the least PPB tolerance with the CE and TLSS garments in the previous study.

These two sets of experiments have shown that subjects, with the appropriate equipment, can tolerate very long periods of high levels of PPB, from both a physiological and discomfort perspective. Given that a number of the subjects felt that they could have continued PPB well beyond the 20 minute limit of these experiments, we have yet to determine the physiological limit to PPB exposure.

It was apparent that the limitations to extended stays at extreme altitudes following rapid decompressions, or even longer exposures to more moderate altitudes, was not limited by the cardiovascular consequences or discomfort of PPB. However, it remained a question whether the improved designs of anti-G ensembles, are of optimal design for providing maximum altitude protection.

In addition to the ground-level experiments on the cardiovascular experiments, over the last 20 years DCIEM has undertaken a series of experiments examining the ability of life-support systems, not originally designed for extreme altitude protection, to provide sufficient oxygen supply, as well as sufficient cardio-vascular support against the adverse effects of PPB, following explosive decompressions up to 80,000 ft. (Holness, *et al.,* 1981; Buick and Porlier, 1991; Fraser, and Ackles, 1994). These experiments have almost all been with subjects wearing the CF partial-pressure jerkin, along with various styles of G-suits. The CSU 15, the RAF Mark 7, and the ATAGS, and EAGLE lower body garments have all been used in these experiments. All of the experiments have been undertaken with a G-suit/PPB pressure ratio of 4:1.

These experiments have involved explosively decompressing well trained subjects from an altitudes of 25,000 ft to 60,000 and 72, 000 ft , or from 35,000 ft to 72,000 and 80,000 ft. As a result we have developed a database of oxygen saturations following rapid decompression. In these experiments oxygen saturations have been monitored with either an HP 47201A ear oximeter, or an Radiometre pulse ear/finger oximeter.

In these experiments subjects breathed 100% oxygen at ground-level for at least 1 hour prior to commencing the experiment to minimize the risk of decompression sickness. Chamber pressure was reduced from

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ground-level to an altitude equivalent of 22,500 ft. Control physiological data was collected for 3 minutes at this altitude. Chamber pressure was then decreased to an altitude equivalent of 35,000 ft. and maintained at that level for up to 3 minutes for the passing of gastrointestinal gas. For the 60,000 ft. and some of the 72,000 ft. rapid decompression experiments the chamber was then recompressed to 22,500 ft. and the subject was rehearsed on the rapid decompression procedure. The subject was instructed to exhale during a 5 second countdown, and rapid decompression of the chamber occurred with the chamber going from 22,500 ft. to 60,000 ft. or 72,000 ft. Rapid decompression was completed in less than 0.6 s. Inflation of the G-suit and PPB was initiated during the rapid decompression. For some of the 72,000 ft. and all of the 80,000 ft. decompressions subjects decompressed from 35,000 ft.

Tables 1, 2, and 3 show the average changes in oxygen saturation following the rapid decompression for a number of experiments extending over 20 years, utilizing different anti-G suits and upper body counterpressure jerkins. All data is presented as changes during rapid decompression with respect to the saturation levels prior to rapid decompression.

Table 1. Decrease in average oxygen saturation at 1, 2, and 3 minutes following rapid decompression to 60,000 ft. with 60 mmHg, 100% O_2 positive pressure breathing, and 240 mmHg anti-G suit pressure.

G-suit	Jerkin	1 min	2 min	3 min
RAF-Mk7	RAF-Mk5	18	21	23
RAF-Mk7	RAF-Mk5	19	21	26
RAF-Mk7	RAF-Mk5/CF	17	18	19
CSU 15	CF jerkin	19	23	25
TLSS	TLSS	25	27	30

Table 2. Decrease in average oxygen saturation at 1, 2, and 3 minutes following rapid decompression to 60,000 ft. with 70 mmHg, 100% O_2 positive pressure breathing, and 280 mmHg anti-G suit pressure.

G-suit	Jerkin	1 min	2 min	3 min
CSU 15	CF jerkin	16	18	20
TLSS	TLSS	18	21	18
ATAGS	CF jerkin	17	21	21
EAGLE	CF jerkin/CF	18	24	25

In the CE/TLSS comparison study (last two rows of Table 1) there was a statistically significant difference in the oxygen saturation with the TLSS and CE garments during rapid decompression to 60,000 ft., with the SaO2 falling further with the TLSS garment. In the comparison study with 70 mmHg PPB (rows 2,3, and 4 of Table 2), SaO2 was maintained to a better degree with TLSS than with ATAGS or EAGLE. The saturation difference

between the three systems was only 6% SaO₂ at the end of the three minutes of exposure to 60,000 ft.; however, this could be critical at these levels of hypoxia and cardiovascular stress.

Table 3. Decrease in average oxygen saturation at 1, and 2 minutes following rapid decompression to 72,000 ft. with 80 mmHg, 100% O₂ positive pressure breathing, and 320 mmHg anti-G suit pressure.

G-suit	Jerkin	1 min	2 min
CSU 15	CF jerkin	16	18
TLSS	TLSS	18	21
EAGLE	CF jerkin	21	31
ATAGS	CF jerkin/CF	35	

At 72,000 ft. the differences between the SaO₂ with the ATAGS and EAGLE garments were more substantial, with a very rapid drop in the SaO₂ following decompression when the subjects wore the ATAGS garment. The differences in SaO₂ may be due to the interaction between the different levels of lower body bladder coverages and the known effects of PPB on V/Q relationships in the lung (Stolp, et al., 1994). The inability of five of the six subjects to complete their ATAGS runs at 72,000 ft. due to this extremely rapid fall in SaO₂ would indicate that the ATAGS garment is not suitable for providing emergency protection at this extreme altitude.

Conclusions and Recommendations

With the use of chest counterpressure, extended coverage anti-G suits, and a 4:1 G-suit to PPB pressure ratio, well trained subjects can tolerate well over 10 minutes, and as much as 20 minutes, of high levels of positive pressure breathing. The ability of subjects to tolerate these PPB exposures has considerable operational impact with respect to mission completion following loss of cabin pressure at altitudes above 50,000 ft.

Our experiments suggest that the use of the extended coverage anti-G suit during PPB and extreme altitude exposures, may result in less protection against hypoxia, than the use of PPB with the older design anti-G suits. One incidence that occurred during our experiments exemplifies the complexity of the problem. One of our subjects lost consciousness during a rapid decompression exposure while wearing the CE ensemble. Her oxygen saturation was better maintained than with the TLSS garment, however, the moderate hypoxia combined with the more severe cardiovascular decrement of the CE system resulted in syncope. In her exposure with the TLSS garment, her oxygen saturation fell to a lower value than with the CE system, but her cardiovascular function was well maintained, and no loss of consciousness occurred.

We have determined that PPB duration can be extended well beyond 10 minutes. There may be considerable utility in using PPB to increase internal body pressure and thus reduce the risk of decompression sickness following loss of canopy pressure at altitudes where hypoxia is not the most significant problem. Bubble formation and growth is strongly dependent on the hydrostatic pressure of the fluid in which bubbles can occur (Ward et al., 1982). Any increase in this pressure will reduce the likelihood of nucleation and subsequent bubble growth. At present, with the loss of cockpit pressurization at altitudes above 35,000 ft., pilots will normally breathe 100% O₂ at a pressure of 30 mmHg. For a pilot at 45,000 ft this reduces her effective physiological altitude to 40,000 ft. If instead the pilot breathed oxygen at 80 mmHg, the physiological altitude would then be reduced to less than 34,000 ft. The extended bladder coverage provided by these newer G-suit designs may make this a practical and safe procedure for reducing the risk of decompression sickness and allow for mission completion in spite of a loss of cockpit pressurization.

Additional research is needed to investigate the combined effects of long-duration PPB and moderate hypoxic stress on the cardiovascular system. Given the substantial differences among the ensembles in maintaining SaO_2 at the extreme altitudes, additional work is also required to optimize G-suit/chest counterpressure jerkin design to provide maximal acceleration protection, provide maximum oxygen delivery at altitude, minimize cardiovascular decrements during PPB, and minimize the risk of decompression sickness during long exposures to altitude stress.

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Discussion

DR. HARDING: Since Dr. Ackles has presented this paper on behalf of Dr. Fraser, it's probably unfair to expect Dr. Ackles to answer questions, but would you like to give it a try?

DR. ACKLES: Sure, I can try.

DR. GOODMAN: Just to elaborate on Dr. Ackles' point regarding the suit pressure ratios, we are seeing a reduction in heart rate using these full-coverage suits in association with an over abundance of circulatory preload which might be causing a stagnation in cardiovascular function. The circulatory stagnation could be harmful when you're in an actual hypoxic environment for a period of time. That's why we think the pressure ratio is important, because if your cardiocirculatory function stagnates there may be poor oxygen transport to the tissues at higher altitudes, while at ground level it might be okay.

DR. MOON: I don't think that can be the explanation. You've shown that the full-coverage suits provide better cardiac output and that's where you would expect higher saturation, yet you're actually seeing the reverse. I don't think it's likely to be a cardiac output phenomenon. It must be either related to lung ventilation/perfusion imbalance or you get less hyperventilation with your better suits and maintain more normal PCO_2's and, therefore, a more physiological pH.

COL. STORK: It may also depend on the effect of the uniform compression on the large muscle masses. I would hope that future studies might look at the A-V differences in oxygen saturation so that we can see if it's the change in blood flow in the periphery that's causing the changes.

DR. GOODMAN: Did Dr. Fraser use an ear oximeter?

DR. ACKLES: No, I think it was a finger oximeter. We found exactly identical results using either the ear oximeter or finger oximeter. We considered the arm congestion, but found no difference on the oximeter readings. So we're now using the finger oximeter routinely, because it is much easier to use.

PROF. ERNSTING: You might want to emphasize to the audience that for every mm Hg PCO_2 you drop, you raise your alveolar PO_2 by one mm Hg. So you only need to have moderate differences in hyperventilation and PCO_2, as Dr. Moon was suggesting, to explain those saturation differences. I would have thought that the PCO_2 would be the largest factor influencing the differences in saturation. You must know either the saturation and PCO_2, or saturation and PO_2, to be able to say what's happening in the arterial blood. I know that if you just hyperventilate you can push up your saturation very markedly. It doesn't improve the oxygen delivery to the tissues. I think I'd be very interested to hear from the Duke University group about what they believe their studies have shown in terms of the difference between arterial PCO_2 and end-tidal PCO_2s in pressure breathing. I think for all of us that's a very important practical point as we would not want to do invasive arterial PCO_2s at 55-60,000 feet.

DR. STOLP: That's an important question. Considering the lung ventilation/perfusion mismatch you really need invasive monitoring to truly know what's going on.

MAJ. CAULKINS: You noted that your subjects were comfortable breathing at 80 mm Hg for 10 minutes. If we're talking about an aircrew member doing this in the actual flight environment, what kind of training would be necessary for them to do it comfortably and how frequently will they need the training?

DR. ACKLES: In later experiments using the full-coverage suits and pressure breathing for 20 minutes, the subjects indicated that they could have continued, but would opt not to continue at the high pressure levels. There seems to be a pain threshold that must be overcome during the first two or three minutes and after that it doesn't seem to make much difference. As long as you have good physiological support and your blood pressure doesn't drop, it seems to be irrelevant after the first few minutes.

DR. STOLP: In training people not to hyperventilate while pressure breathing, it is important to know what the blood gases are because if they slow their breathing rate sufficiently they will reduce O2 delivery to the tissues.

MAJ. CAULKINS: In our studies, we found that neck discomfort was the end point rather than blood gases.

DR. ACKLES: I haven't pressure breathed for ten years, but we haven't had many complaints about neck discomfort recently. I used to experience a good bit of neck and arm discomfort in early studies at Farnborough, but when we got into the Phoenix studies, it didn't seem to bother us as much and I don't know why.

DR. GOODMAN: We may have become very habituated to the discomfort in the arm and neck area. From the facial viewpoint, the mask's are much better today in distributing the pressure more to the bony surfaces and it seems to make them more comfortable. One thing I would add is that as PBG comes on-line for many air forces in the world, aircrew are going to be training on the panels at ground level and they're going to become much more familiar with pressure breathing than they would have if it were just for altitude protection. So in the next ten years we should see a lot more aircrew very familiar with PPB.

COL. SHAFFSTALL: We can't assume that all pilots will be trained on pressure breathing panels.

DR. GOODMAN: That's a problem.

SQN LDR RYLES: The neck pain has been quite noticeable in our subjects. The impression we've been getting is that levels of 50 and 60 millimeters are suitable for short emergency get-me-down scenario, but any longer than that the subjects are really struggling to continue, especially at the 60 mm Hg level. With experience, the subjects can control hyperventilation and other factors as well as get used to a certain amount of discomfort, but the neck pain still stays. With our first subject we didn't manage many minutes at 60 mm Hg, largely because we had gone from 50 to 60 mm Hg in a short time interval. We found that we had to leave a couple of days between 50 and 60 mm Hg exposures for the neck discomfort to settle down. Very often there would be a slight hoarseness of voice as well and other factors indicating some of the trauma had resulted from the high PPB.

DR. PILMANIS: Is there much difference between positive pressure breathing 60 mm Hg at ground level versus altitude from the standpoint of either comfort or any other aspect?

DR. BOMAR: Pressure breathing is relatively more comfortable at altitude. There is also a certain amount of anxiety associated with being decompressed to 60,000 feet. It's a whole different situation than at ground level where the mask pressure is just dialed to higher levels. You find that you can easily control your breathing at ground level. It's another experience to rapidly decompress to 60,000 feet, but it's easier to breathe using the current pressure assembly. In fact, our subjects preferred being exposed to 60,000 feet using the mask/vest/G-suit ensemble than to 50,000 feet using just a mask and helmet.

DR. PILMANIS: Is it significantly easier, so that ground-level training might not truly represent the high-altitude situation?

DR. BOMAR: I suppose you could train at ground level, but I think that the altitude experience is probably better.

LT. COL. DEMITRY: If there's a significant difference, would it be possible to conduct ground-level training that might better simulate the altitude condition? Could a training adjustment be made that is a logical and inexpensive way to train crew on the ground? On the other hand, after experiencing one flight in a U-2, I can tell you the training that you get inflight is very, very different than anything you get on the ground. From a training perspective, the apprehension is something that might be very valuable to aircrews.

DR. STOLP: The compressibility of the gas is different. You might want to considering using a helium mixture to mimic the compressibility factor.

LT. COL. DEMITRY: That's another way of doing it.

DR. STOLP: It would be an alternate way. The distribution of the pressure to the lungs may be affected by the different breathing patterns.

COL. STORK: We might be able to demonstrate the physiological effects during ground level , but that's not nearly as valuable to the crew member as demonstrating the effects of his equipment at altitude where he or she is going to have to use it. So I think training must include exposure to altitude similar to that conducted in the UK.

DR. ACKLES: When we were involved in the TLSS program I tried and failed to persuade the USAF, as a final test, to actually take the TLSS system into an F-15 and decompress the aircraft at 60,000 feet, even though we had already done it successfully at 54,000 feet with our F-104. I thought that's the ultimate way to prove to the aircrew that you have confidence in the protection system.

PROF. ERNSTING: We simulate the pressure breathing time curve we're going to get at altitude during ground-level training and, almost without exception in vast numbers of aircrew, they indicate it's much easier at altitude, provided they don't get abdominal gas pain. That may be partly from the relief over having completed the chamber run. Everybody is slightly euphoric after being at 60,000 feet in an altitude chamber. Our training frequency is every two years or so, depending on whether the aircrew still requires high-altitude instruction.

DR. GOODMAN: Our subjects experience the same thing. All of them say it just felt easier to repeat the pressure run and they'd much rather experience PPB in the chamber. Everyone hates the pressure panel during ground training. Everyone, including myself, would much rather be exposed to G with 70 mm Hg in the mask than on the pressure breathing panel at 70 mm Hg. Part of this feeling may be that the altitude exposure more closely simulates the flight environment. So we need the same specificity for the PBA as we do for PBG training.

Development of USAF Pressure Breathing Systems

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Background

The COMBAT EDGE (Combined Advanced Technology Enhanced Design G-Ensemble) positive pressure breathing system for +Gz protection currently used in USAF fighter aircraft was initially developed in a USAF program called the Tactical Life-Support System (TLSS). TLSS was the first USAF 6.3 (Advanced Development) life-support program. It was a highly ambitious effort which had design requirements to provide high-altitude protection, improved G-protection, personal cooling, and improved nuclear, biological and chemical (NBC) protection. In addition, the TLSS program integrated nuclear and laser eye protection, and onboard oxygen generation into the life-support system. While the TLSS concept of combining all of these design requirements into a single system did not reach an operational status, many of the TLSS components and designs were used in either operational or future equipment designs.

Two of the major TLSS efforts, improved G-protection and altitude protection to 60,000 ft., involved the use of assisted PPB (Positive-Pressure Breathing via an oral-nasal mask assisted by a counterpressure garment covering the chest or torso). This paper describes the development of TLSS positive pressure breathing systems and their impact on current systems.

TLSS Program Development

Prior to the TLSS program, the life-support equipment development and acquisition process did not have an intermediate development step to move successful Exploratory Development (6.2) efforts into an Engineering and Manufacturing Development (6.4) program that could provide improved equipment to the aircrew (Table 1). As the initial life-support advanced development effort for the USAF, the TLSS program established the basic methodology by which equipment could be made flightworthy and be flight tested. To meet this objective, the TLSS system developed and integrated the items shown in Table 2 into a flightworthy life-support system.

Table 1. USAF acquisition process and funding category designations.

BASIC RESEARCH	6.1
EXPLORATORY DEVELOPMENT	6.2
ADVANCED DEVELOPMENT	6.3
ENGINEERING AND MANUFACTURING DEVELOPMENT	6.4

Table 2. Tactical Life-Support System.

HIGH-ALTITUDE PROTECTION TO 60,000 Ft.
PRESSURE BREATHING FOR G PROTECTION TO +9Gz
NBC PROTECTION FOR LIQUID AND VAPOR THREATS
PERSONAL COOLING USING A LIQUID COOLING SYSTEM
NUCLEAR FLASH PROTECTION (PLZT VISOR)
LASER PROTECTION (TINTED VISOR)
ON-BOARD OXYGEN GENERATION SYSTEM (OBOGS)

Since the objective of TLSS was to demonstrate flightworthy hardware in an integrated system, a number of new equipment designs were required to accomplish this task. A new breathing regulator was developed to use the gas generated by an on-board oxygen generation system (OBOGS), meet the system breathing requirements and provide PPB for both altitude and G-protection. The oxygen supply provided for the flightworthy system also had to incorporate emergency and backup oxygen for bailout and OBOGS failure. An electronic G-valve was required to provide rapid G-suit inflation and to provide acceleration sensing for the breathing regulator. In addition, a seat-mounted personal equipment connector was developed to provide a single point quick disconnect of all man-side pneumatic and electrical connections. The man-side equipment was also entirely new and included an integrated upper pressure garment and an extended coverage anti-G suit. Included in the new man-side equipment was a light-weight helmet with a mask tensioning bladder and an advanced design oxygen mask and retention system capable of containing high levels of PPB during +9Gz accelerations. These new equipment items are only those that relate to the oxygen delivery system. The development of new equipment to meet the NBC, personal cooling, nuclear flash blindness, and laser eye protection requirements added even more complexity and engineering challenges to the TLSS effort.

Table 3. TLSS flight test results.

FULLY INTEGRATED TLSS TESTED AT EDWARDS AFB IN 1986-1987

- SYSTEM INSTALLED IN THE FRONT SEAT OF AN F-15B
      - 26 SORTIES INCLUDED FAMILIARIZATION, HIGH-ALTITUDE, AIR-AIR AND   AIR-GROUND
      - UNQUALIFIED SUCCESS

SIMPLIFIED SYSTEM TESTED AT EDWARDS IN 1987

- REAR SEAT F-16B, 24 SORTIES, MOSTLY AIR-AIR
      - USED THE AIRCRAFT LOX SYSTEM
      - MODIFIED CRU-73 REGULATOR
      - EXISTING (HIGH-FLOW) VALVE

PILOT EVALUATIONS WERE HIGHLY FAVORABLE, STRONG RECOMMENDATIONS FOR OPERATIONAL DEVELOPMENT

Considering the complexity of the new system and the very demanding flight test scenarios, the flight tests that were conducted at Edwards AFB, CA in 1986 and 1987 were an unqualified success (Table 3). The system was installed in the front seat of an F-15B and 4 test pilots flew a total of 26 sorties using the TLSS system. Test flights included high- and low-altitude familiarization flights, a 28,000 ft. rapid decompression flight, a 60,000 ft. flight and simulated air-to-air and air-to-ground missions. The aircraft cockpit was not decompressed during the 60,000 ft. flight; however, the altitude pressure breathing system was thoroughly evaluated in the altitude chamber. The pressure breathing system for altitude provided safety pressure between 25,000 - 39,000 ft. Between 39,000 - 60,000 ft., the pressure schedule increased to reach a maximum mask/torso garment pressure of 70 mmHg. In the altitude protection mode, the G-suit was pressurized at 4 times the breathing pressure at altitudes above 39,000 ft.

While all of the components of the TLSS functioned well, the test pilots gave their highest ratings to the improved acceleration support. In an additional flight test effort, several simplified systems to provide positive-pressure breathing for G-protection (PBG) were developed and flown in an F-16B. One of the PBG systems tested in the F-16 made use of a CRU-73 breathing regulator that was modified to provide pressure at G. The acceleration cue for the modified regulator was accomplished by running a pressure sensing line from a mechanical high-flow G-valve to the regulator. TLSS-derived man-side equipment was used to evaluate the simplified PBG system. The PBG pressurization schedule for both the F-15 and F-16 flight tests was 12 mmHg/G starting at +4Gz. This schedule provided a maximum pressure of 60 mmHg at +9Gz. Coupled with the success of the TLSS acceleration protection system, the events listed on Table 4 triggered a revision in the program and directed its focus toward G-protection.

Table 4. Pressure breathing for G (PBG) program drivers.

> **INCREASE IN G-INDUCED LOSS OF CONSCIOUSNESS IN 1988 GAVE URGENCY TO PBG PROGRAM DEVELOPMENT**
>
> **- ONE ACCIDENT BOARD INTERVIEWED A FORMER TLSS TEST PILOT AND RECOMMENDED PBG DEVELOPMENT**
>
> **IN APR 88, HQ TAC REQUESTED EARLIEST POSSIBLE DEVELOPMENT OF A RETROFITTABLE PBG SYSTEM**
>
> **- PBG REQUIREMENTS: OPTIONAL, TRANSPARENT, MAKING MAXIMUM USE OF EXISTING EQUIPMENT**
>
> **- TASKING EFFECTIVELY SPLIT PBG OUT OF THE TLSS PROGRAM AND IS NOW KNOWN AS COMBAT EDGE**

Since TLSS included a number of items which could provide improved G-protection, a centrifuge study was conducted to determine the optimum design that could be provided on a cost and time effective basis (Table 5).

Six well trained centrifuge subjects were used to evaluate the five experimental conditions shown on Table 6. While each of the experimental conditions used the TLSS mask and helmet, three different types of torso counterpressure garments were evaluated. The TLSS suit (Ensemble I) has the torso counterpressure bladder and anti-G suit integrated in a single garment. The TLSS G-suit bladder covered an approximately 30% larger area of the lower body than the standard CSU-13B/P anti-G suit. The torso area covered by chest counterpressure bladder of the integrated TLSS suit and the bladder of the TLSS component vest (Ensemble II) were approximately the same with the TLSS component vest being worn with a CSU-13B/P anti-G suit. While the bladder of the extended pressure vest (Ensembles III, IV and V) covered approximately the same torso area as the TLSS garment, the extended vest had a longer "shirt tail" that allowed the bottom of the extended vest to be tucked in and worn under the abdominal bladder of the anti-G suit. Ensembles IV and V used the extended vest with a full-coverage anti-G suit. The full-coverage anti-G suit used in this evaluation provided uniform circumferential pressure coverage to

the subjects' legs, feet and abdomen. The modified CRU-73 breathing regulator was used for Ensembles I - IV. Ensemble V used an experimental breathing regulator fabricated by the Normalair Garrett Ltd. NGL Co., Yoevil, U.K. which offered the same PBG schedule as the modified CRU-73, but had significantly less breathing resistance. The primary measure of merit used in this study was the subject's endurance time on a 5-9 G Simulated Aerial Combat Maneuver (SACM) acceleration profile.

Table 5. Evaluation of PBG components.

IN ADDITION TO PBG, TLSS INCLUDED SEVERAL DESIGN CONSIDERATIONS WHICH LIKELY CONTRIBUTED TO IMPROVED ACCELERATION PROTECTION:

- INTEGRATED GARMENT
- ENHANCED G-SUIT COVERAGE
- VERY LOW-RESISTANCE BREATHING SYSTEM

TO WHAT EXTENT WOULD MODULARIZING THE SYSTEM AND USING EXISTING EQUIPMENT POTENTIALLY COMPROMISE TLSS G-PROTECTION?

TLSS G-PROTECTION COMPONENTS EVALUATION, STUDY TO EVALUATE THE INFLUENCE OF:

- MODULAR OVERGARMENTS (G-SUIT & VEST)
- EXTENT OF G-SUIT COVERAGE (CSU-13B/P AND FULL-COVERAGE SUIT)
- BREATHING RESISTANCE (MODIFIED CRU-73 AND LOW-RESISTANCE REGULATOR)

Table 6. TLSS component evaluation.

COMPARED FOUR CANDIDATE EQUIPMENT COMBINATIONS TO THE TLSS INTEGRATED ENSEMBLE			
ENSEMBLE NUMBER	TORSO GARMENT	ANTI-G SUIT	BREATHING REGULATOR
I	TLSS INTEGRATED PRESSURE VEST	TLSS EXTENDED COVERAGE SUIT	MOD CRU-73
II	TLSS COMPONENT PRESSURE VEST	CSU-13B/P	MOD CRU-73
III	EXTENDED PRESSURE VEST	CSU-13B/P	MOD CRU-73
IV	EXTENDED PRESSURE VEST	FULL-COVERAGE SUIT	MOD CRU-73
V	EXTENDED PRESSURE VEST	FULL-COVERAGE SUIT	NGL, LOW-BREATHING RESISTANCE
ALL ENSEMBLES USED THE TLSS OXYGEN MASK AND HELMET			
SIX SUBJECTS RODE EACH ENSEMBLE ON THE 5-9 G SACM PROFILE TO EXHAUSTION USING THE F-16 SEAT CONFIGURATION			

Combat Edge Development

Based on the conclusions drawn from the component evaluation (Tables 7 and 8) and the TLSS flight evaluations, the COMBAT EDGE system was established as an Engineering and Manufacturing Development (6.4) program. The focus of this program was to provide PBG while minimizing the equipment changes required to implement the system.

Table 7. Results from the TLSS Component Evaluation: Mean SACM Tolerance Time (Seconds) and Standard Deviations.

ENSEMBLE	SACM TOLERANCE TIME ± STANDARD DEVIATION
I	137 ± 34s
II	128 ± 30s
III	127 ± 21s
IV	200 ± 30s*
V	237 ± 38s*

*$p < .01$ via ANOVA

Table 8. Conclusions drawn from the TLSS Component Evaluation.

NO EFFECT OF MODULARIZATION

- COMBINATION III WAS CHOSEN AS THE COMBAT EDGE BASELINE

THE FULL-COVERAGE G-SUIT GREATLY INCREASED TOLERANCE
- THE FULL-COVERAGE G-SUIT, NOW KNOWN AS THE ADVANCED TECHNOLOGY ANTI-G SUIT (ATAGS) WOULD CONTINUE AS A DEVELOPMENTAL EFFORT

- IF SUCCESSFUL ATAGS WOULD EVENTUALLY REPLACE THE CSU-13B/P

LOW-BREATHING RESISTANCE WAS ALSO IMPORTANT

- THE COMBAT EDGE REGULATOR SPEC. CHANGED TO INCLUDE HIGH-FLOW CAPACITY

Table 9. Combat Edge Prototype Equipment Used for Man-Rating and Operational Test and Evaluation.

ENGINEERING AND MANUFACTURING DEVELOPMENT SYSTEM DELIVERED IN THE SUMMER OF 1989 WAS COMPOSED OF:

- CRU-93 OXYGEN REGULATOR (NO LONGER MODIFIED CRU-73)

- NEW HIGH-FLOW G-VALVE (TIGHTER SPECIFICATIONS)

- MBU-20/P OXYGEN MASK

- MODIFIED HGU-55/P HELMET

- NEW COUNTERPRESSURE VEST (DERIVED FROM THE EXTENDED VEST)

- CONVENTIONAL CSU-13B/P ANTI-G SUIT

COMBAT EDGE WAS TARGETED FOR BOTH F-15 AND F-16

Summary

The COMBAT EDGE system was successfully developed and operational testing was completed in 1992 (Table 9). In April 1995, the retrofit of COMBAT EDGE for all USAF F-16s was completed and the system was in use on a routine basis for missions requiring high levels of G-protection (Table 10).

Table 10. Combat Edge Status.

MAN RATING COMPLETED IN 1990

OT&E COMPLETED IN 1991

- SYSTEM ACCEPTED IN F-16s FOR AN EXTENDED FOLLOW-ON OPERATIONAL VALIDATION, COMPLETED IN 1993

- F-15 AIRCRAFT MOD., SUMMER 1995

- F-15 MANSIDE EQUIPMENT, FY 1996

FULL F-16 FLEET RETROFIT COMPLETED IN 1994

ALL NEW F-16s DELIVERED WITH COMBAT EDGE CAPABILITY

F-15E DELIVERED WITH COMBAT EDGE CAPABILITY

As an Advanced Development program, TLSS was not intended to specifically generate new concepts in life-support equipment. The charter of the TLSS effort was to provide a vehicle to incorporate the laboratory generated technical advancements into an integrated system to improve aircrew life support. Concepts advanced by TLSS are being used in current life-support systems and are being evaluated for the next generation of life-support equipment. Specifically, TLSS pressure breathing concepts are flying today in COMBAT EDGE and a PBG system

is planned for the F-22. The TLSS pressure breathing systems for altitude protection did not have an immediate operational requirement in the F-15 and F-16; however, much of the TLSS altitude protection technology can be seen in the F-22 life-support system.

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Discussion

DR. ACKLES: I was involved early on in the TLSS program and feel the main reason that the program received the early high-level attention from the Air Force was that the F-15 pilots wearing the TLSS ensemble invariably beat the F-16 pilots one on one, even though the F-16 is a more agile aircraft. As an aside, I'm still a supporter of an integrated garment. I don't understand how anyone can say that it's more comfortable to place other garments over the standard flight suit, considering the wrinkling and crumpling that occurs under the assembly. It would seem more appropriate to remove your standard flight suit and slip into an integrated assembly that is appropriate to the mission needs. I think the people who have flown the integrated suits, the test pilots, have thought it was fine. It's been my experience in meetings like this, that qualitative decisions were made that the integrated ensemble, with hoses hanging out, would not be pilot acceptable, i.e., rather than conducting a proper trial. You might find that it would be more acceptable rather that the current modular approach.

COL. STORK: The discussion always deteriorated in those days to whether the pilot would be allowed to wear it to the club for lunch.

MAJ. NEUBECK: We've got airplanes that are performing better and can sustain higher Gs longer. I was in the 422nd Squadron at Nellis when they were using COMBAT EDGE and the guys were commenting that they were less fatigued wearing it because you do not have to strain as hard or you're not having to work as hard to maintain the higher Gs.

DR. ACKLES: I agree. It's just the general discomfort of having a double suit. Then you need to consider the use of Gortex to decrease the thermal discomfort. This won't alleviate the situation since you have so many layers of insulation.

MAJ. NEUBECK: If you give a pilot a full-coverage suit and indicate that he could pull 8 Gs at rest, he'd probably be glad to get it, because the high-G environment is very stressful.

COL. HILL: We have to ask the question why the TLSS wasn't bought by the United States Air Force. When we started losing airplanes at a much greater rate from exposure to high G, the using community asked the acquisition community how long it would take to complete development and produce TLSS, and the answer was too long and it cost too much. So we separated out the G protection portion of the program, expecting full well that later on we'd go back and integrate the total system. That's where we've fallen apart because now cost is our driver and we would have to replace everything in the cockpit and that's unacceptable.

DR. ACKLES: Yes, I realize that.

COL. HILL: I know you do but the some of the people here weren't that close to the program.

Pressure Breathing and Acceleration Atelectasis

Ulf I. Balldin, M.D., Ph.D.

Acceleration induced pulmonary atelectasis (among pilots described as G-cough) is a condition that may affect fighter pilots wearing an anti-G suit and breathing oxygen during and after pulling high G-loads (12, 13, 14, 16). A gas mixture consisting of about 70 % or more oxygen may be enough to provoke the symptoms (Figure 1).
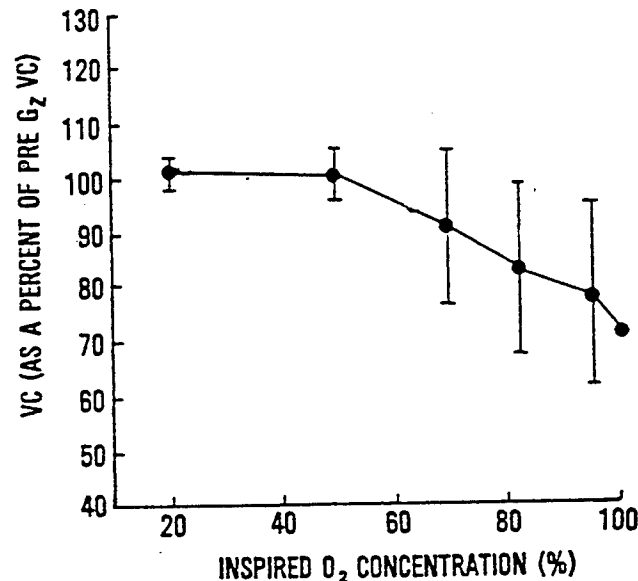

Figure 1. Vital capacity measurements after simulated aerial combat maneuver G-profiles with different inspired oxygen concentrations. After Haswell et al. (12) with permission from Aviat. Space Environ. Med.

The G-load need not be very high to induce this phenomenon, only 3 to 4.5 G may be required for only a short time. The symptoms consist of throat irritation, chest pain, coughing, and shortness of breath. The symptoms may persist for only a few minutes, but may also last for several hours. Deep breaths after the flights may alleviate the symptoms. Vital capacity measurements may initially provoke coughing, but repeat measurements diminishes the symptoms and also increases the lung volumes by each measurement procedure (Figure 2).

The symptoms are accompanied by reduced pulmonary vital capacity measurements and signs of atelectasis on pulmonary X-rays (10). The atelectasis is due to absorption of trapped oxygen with closed off airways resulting in the collapse of the alveoli in the dependent lung during the increased G-load (9). The airways may close due to external compression by the increase weight of the dependent lung and due to the action of the abdominal bladder of the anti-G suit by raising the diaphragm. The formation of airway mucosal edema due to blood congestion in the dependent lungs during increased G-loads may also contribute. Surface tension forces in the alveoli may then suffice to maintain the collapse of the lung. Measurements of reductions of vital capacities of up to 60% have been recorded (17), although such big reductions suggested to be called labile atelectases might partially be due to reflex inhibition of inhalation due to irritation of the airways (9). The smaller volume reductions of the more stabile atelectases seem to be closer to the atelectasis-equivalent findings on X-rays.

Similar phenomena may also be seen in divers breathing oxygen for longer periods (Figure 3). Just immersion with the head above water may induce decreases in the measured lung vital capacity of up to 42 % (4,

6). The airway closure induced by the negative pressure breathing and reduced lung volume during head-out immersion also causes pulmonary absorption atelectasis, when the breathing gas is oxygen. When air is breathed during head-out immersion--creating a negative pressure breathing condition--such atelectasis formation is not created (4).
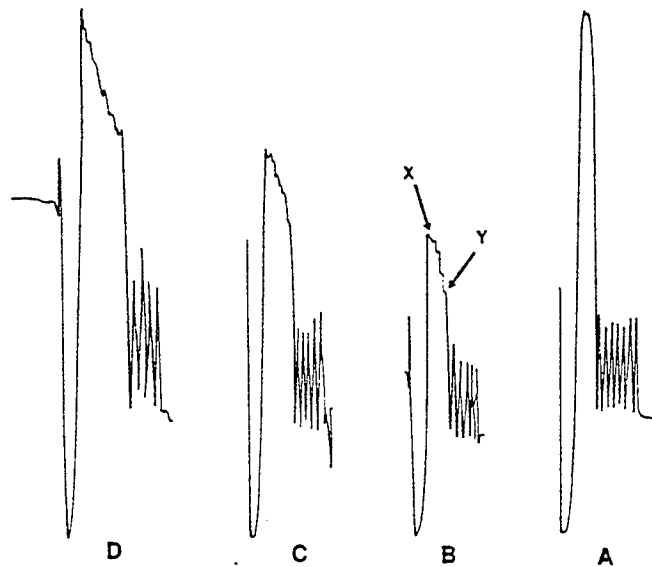
Figure 2. Vital capacity measurements taken before (A) and after the centrifuge ride (B, C, D with 1 min interval). Y represents labile atelectasis and X stabile atelectasis. From Tacker et al. (17) with permission from Aviat. Space Environ. Med. (Tracings are read from right to left.)

When flying at higher altitudes with lower cabin pressure, the need for higher oxygen concentration to avoid hypoxia and the fewer gas molecules per tidal volume of breathing gas, the likelihood of developing absorption acceleration atelectasis should be higher. On the other hand, at such high altitudes, the fighter aircraft are less likely to pull high Gs, thus counteracting this increased tendency to atelectasis formation.

Figure 3. Spirogram in an oxygen breathing subject (without positive pressure breathing) during head-out immersion (from Dahlback and Balldin (6) with permission from Undersea Biomedical Research).

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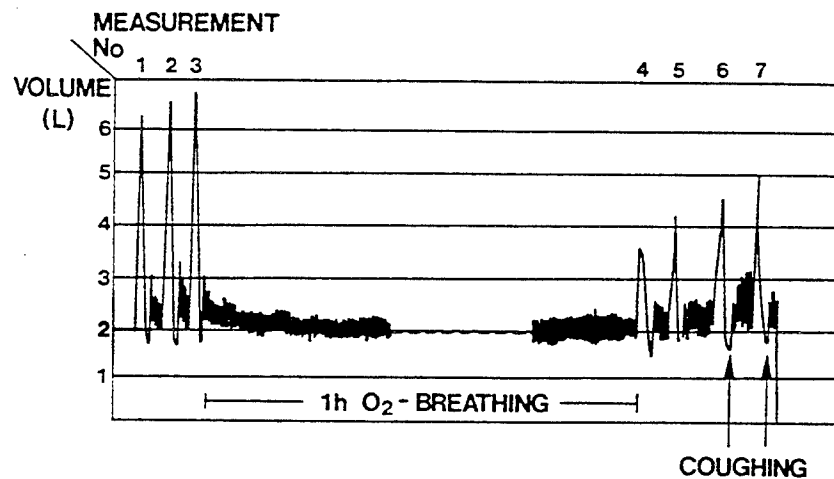Admixture of 5% argon, which is a byproduct from On Board Oxygen Generating System (OBOGS), to a breathing gas consisting of pure oxygen, does not reduce the magnitude of acceleration atelectasis nor the severity of the lung symptoms (12). When more than 70% of the breathing gas is oxygen, independent of whether the inert gas is nitrogen or argon, acceleration atelectasis may be formed. Occasionally, such symptoms may occur even with oxygen concentrations as low as 50% (12), but with less oxygen and during air breathing the symptoms do not appear.

For many years, positive pressure breathing has been used during rapid decompressions of aircraft as a method of avoiding hypoxia (8). Usually pressure breathing is delivered when the cabin altitude exceeds 40,000 ft (12,192 m), with breathing pressure increasing to 30-35 mm Hg (4-4.6 kPa) at 50 000 ft (15,240 m). For "get me down" capability from even higher altitudes, emergency positive pressure breathing with breathing pressures of 50 mm Hg (6.7 kPa), 70 mm Hg (9.3 kPa) or still higher, may be used for short periods. In these cases a counterpressure to the thorax is applied by a vest with air bladders administering the same pressure as in the airways.

Many years ago, the Swedish Air Force and Defence Materiel Administration developed a so called two-pressure flying suit system for "get me down" purpose in case of an explosive or rapid decompression. This system used 50 (6.7 kPa) and 70 mm Hg (9.3 kPa) positive pressure breathing with similar counterpressure to the thorax in a bladder of an integrated vest. For minimizing breathing gas leakage from the breathing mask, a neck mounted gas bladder in the helmet was added, which was inflated with the same pressure as the breathing pressure in order to tighten the mask to the face. With 50 mm Hg this system was successfully tested in explosive decompressions in less than 0.5 s in an altitude chamber from 9000 m (29,500 ft) to 17,500 m (57,400 ft) and with 70 mm Hg to 20,000 m (65,600 ft) (1). To counteract the hemodynamic adverse effects of positive pressure breathing, such as blood pooling in dependent body regions accompanied by a tendency for cerebral blood pressure to drop, a pressure in the anti-G suit was applied about 3.2 times the breathing pressure.

Positive pressure breathing has also been introduced to increase the endurance tolerance to high G-loads (5), preferably in combination with counterpressure to the thorax by vest bladders, so-called balanced pressure breathing during G (PBG). Such a system (e.g., COMBAT EDGE) is now in operational use in USAF F16 aircraft. This system gives a linear increase of the breathing pressure from +4 Gz to a maximum of about 60 mm Hg (8 kPa) at +9 Gz with the same counterpressure in the thoracic bladder in the vest. Other Air Forces are following, e.g., the Tactical Flight Combat Suit developed for the Swedish Air Force Gripen fighter aircraft. With this system the breathing pressure starts from a safety pressure of about 3 mm Hg (0.4 kPa) at +4 Gz linearly to about 50 mm Hg (6.7 kPa) at +9 Gz. The French Air Force is currently testing a maximum breathing pressure of about 70 mm Hg (9.3 kPa) for G-protection. This system has been shown to substantially increase especially the endurance G-tolerance of fighter pilots (2).

Positive pressure breathing has been shown to counteract and to reduce or eliminate absorption atelectasis during several conditions. Thus, the pulmonary atelectasis induced by longer periods of oxygen breathing during head-out immersion may be totally eliminated by introducing positive pressure breathing of about 15-25 cm H2O (6). Similarly, when introducing a closed circuit oxygen breathing apparatus, with the breathing bag on the dorsum of the diver, creating about -2 kPa (-20 cm H2O) static lung load (corresponding to a negative pressure breathing) pulmonary atelectasis may develop (7). When the breathing bag was mounted on the chest of the diver, creating about +1 to +2 kPa (10-20 cm H2O) static lung load (corresponding to a positive pressure breathing) the risk of pulmonary atelectasis was to a great extent eliminated.

During high G-loads positive pressure breathing has also been shown to reduce or eliminate the G-induced pulmonary atelectases during oxygen breathing. Thus, when exposing subjects in a centrifuge to simulated aerial combat maneuvers up to either +4.5 or +9 Gz, while breathing with 30 mmHg (4 kPa) unassisted positive pressure breathing both atelectasis symptoms and vital capacity reductions were diminished or eliminated (17).

The breathing regulator must allow a sufficient breathing flow so that it is not creating a negative pressure during inhalation, which would tend to increase the risk of atelectasis. Old breathing regulators have a tendency to restrict breathing flow creating negative mask and airway pressures, when the pilots require a high breathing

volume executing their straining maneuvers at high G-loads. In most cases, this will be taken care of by pressure breathing during G.

When breathing air during G-loads, the arterial oxygen saturation is decreased with time (3, 12, Figure 4). With oxygen breathing, the desaturation tends to be much less, despite the increased risk of pulmonary atelectasis and pulmonary shunting of blood (12).

Therefore, there should not be any major decrements in pilot performance with oxygen breathing, except for that caused by the symptoms and inconvenience of atelectasis. Pressure breathing during G, though, should help to alleviate most of the still existing minor negative effects of oxygen breathing. The ventilation/perfusion ratio is changed during positive pressure breathing and some of the shunts will be decreased (15). The oxygen desaturation is also less with pressure breathing than without (5, 15), suggesting an improvement in ventilation/perfusion ratio, similar to the improvements seen in the oxygenation in patients when applying continuous positive airway pressure (CPAP) in the respirator.

Respiratory and muscle straining maneuvers necessary to maintain a sufficient cerebral blood pressure and flow during high G-loads also tend to diminish the pulmonary atelectasis formation (17). The mechanisms for this are more difficult to explain than with unassisted positive pressure breathing, as both airways and intrapleural pressure will be increased simultaneously. The deep inspirations during these maneuvers may open up some of the closed or nearly closed airways.

Inspired O_2 (percent)	21%	50%	70%	82.5%	95%	100%
S_aO_2	87.5	91.6	95.0	95.8	96.6	97.8
	±4.28	±4.42	±2.78	±2.43	±5.18	±2.60
n( )	(7)	(9)	(8)	(8)	(6)	(12)

Figure 4. Effect of increasing inspired oxygen concentration on arterial oxygen saturation (S_aO_2) during simulated aerial combat maneuver G-profiles. From Haswell et al. (12) with permission from Aviat. Space Environ. Med.).

The amount of atelectasis formation with balanced or assisted positive pressure breathing during increased G-loads has not been evaluated yet. The condition is in some respects similar to when doing the straining maneuvers with both increased airway pressure and intrapleural pressure, where the pulmonary atelectasis formation seem to be avoided (17). The increased pulmonary ventilation and increased tidal volumes with assisted pressure breathing during G-loads (5) would also act to decrease the tendency to atelectasis formation. Preliminary information by Green et al (11), though, has shown that vital capacity and expiratory reserve volume are reduced during G, but no apparent difference was found with normal breathing and pressure breathing either with counterpressure to the thorax or not. In a survey of 49 fighter pilots by Travis and Morgan (18) the only adverse events found with the COMBAT EDGE positive pressure breathing anti-G suit system using counterpressure to the thorax in the F16 pilots was an increased incidence of dry cough. This could, however, be attributed to the extremely dry conditions at the test site for this group of pilots compared to the control group, who flew in a much more humid environment. No other symptoms that could be attributed to G-induced atelectasis were found. The pilots were using the F16 normal breathing mixture at the flown altitude, mostly consisting of not more than 60 % oxygen, except for the very short periods at high G. During these short periods the pressure breathing oxygen injection system administers up to about 85 % oxygen and then returns to less than about 60% depending upon the cabin altitude of the aircraft. In F18 aircraft with the Naval EAGLE balanced positive breathing equipment using

100 % oxygen for the entire flight time, sporadic reports of symptoms of G-induced lung atelectasis have occurred (T. Travis, personal communication).

Thus, the incidence of G-induced pulmonary atelectasis using balanced pressure breathing during G with more than 70 % oxygen has to be determined. At the National Defence Research Establishment in Sweden, a technique using Respitrace with elastic electric bands to register the relative chest volumes and, thus, to get an estimate of the relative lung volumes at different lung regions has been developed after initial substantial difficulties (Gustavsson P and Gronqvist M, personal communication). The advancement in this technique appears to be promising, so that it may be useful in the centrifuge for on-line information of pulmonary mechanics with balanced pressure breathing.

In conclusion, G-induced pulmonary atelectases seem to be a minor problem with less than 70% oxygen in the breathing gas. When using higher oxygen concentrations symptoms may be experienced, but these are counteracted by unassisted pressure breathing during G and by straining maneuvers. If these symptoms will also be seen when breathing 70% or more oxygen accompanied by assisted or balanced pressure breathing during G remains to be investigated.

DR. BALLDIN: I would like to emphasize that, if a fighter pilot really has symptoms of atelectasis, overall performance probably will be negatively influenced. Therefore, I would strongly recommend continued efforts to accomplish research in this area in order to find out if atelectasis formation really exists during operational flying with PPB and if this atelectasis formation is sufficient enough to cause symptoms. If that is the case, it would be important to find measures to eliminate these symptoms.

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Discussion

COL. HILL: Why is there an apparent national difference in the occurrence of acceleration induced atelectasis? I know we've discussed this before, but I think this group would enjoy hearing about it. Any thoughts?

DR. ACKLES: I don't think there is a difference in our research data. Our position is that we don't think there is an operationally significant problem. We believe there is a certain amount of atelectasis. If we conduct the study that Dr. Balldin is suggesting relating to assisted PBG, I think the fit and tightness of the Jerkin will be critical. We have made a policy to tighten the Jerkin to a fully inflated chest. If you tighten it when the chest is in the relaxed state it can be very uncomfortable, especially if you attempt to take a full inspiration. If you tighten the garment too tightly on the chest you could end up with significant atelectasis. At least, that's our current hypothesis. We're going to be doing flight trials in the near future.

PROF. ERNSTING: Our experience came in the late 1970s flying in our Hunter aircraft with unassisted positive pressure breathing at 30 mm Hg. That amount of pressure breathing certainly did not remove the symptoms of acceleration atelectasis. Dr. David Root, an American exchange physician pilot and several of the other pilots, collected sufficient data to cause us to put 40% nitrogen and 60% oxygen in the oxygen cylinders in the airplane. This step required quite a lot of persuasion of the aircraft authorities at Farnborough. This was in the early days of looking at unassisted positive pressure breathing.

I agree entirely with the other point that Dr. Ackles made. It's very dangerous to argue the findings about assisted positive pressure breathing without specifying the equipment you're using. For various reasons in the UK we are still using a bladder system which completely surrounds the chest. Perhaps that's my bias which has influenced people, but I believe you need good respiratory counterpressure at high altitude. We also fit the garments on full inspiration. When the garment is pressurized, there's full pressurization over the chest and it pushes the chest backwards. In our flight experience with pressure breathing under G, with that type of waistcoat and our full-coverage anti-G trousers and 100% oxygen systems, we still have our test pilots and subjects complaining of the symptoms of acceleration atelectasis. We have not made objective measurements before and after flight. We're simply talking symptoms.

So I think it's very much chest position when you're actually being exposed. If you're down below your normal FRC then you're probably going to get quite a lot of atelectasis and get symptoms afterwards. Again, I don't think aeromedically or physiologically, we have any differences across the Atlantic. I believe that it's primarily a difference in the attitudes of our operational staffs who make the final decisions. When we discovered the condition in the early 1950s, we conducted longitudinal studies to see if we could find any long term consequences of atelectasis and we told the staff clearly there were none. They took the position that they were very unhappy to have aircrew coughing, etc., when they had just come from the flight. It came up again when we were building our version of the AV-8B, the Harrier GR5, with the Navy's very high oxygen in their simple molecular sieve oxygen concentrator. And again it was an air staff decision. They sent the chief test pilot and a couple of other test pilots to go and fly in the aircraft at St. Louis and they came back saying they were getting symptoms of atelectasis and that led to spending a lot of money to introduce a system which would ensure that up to 15,000 feet aircraft altitude we don't get more than 60% oxygen. So that's the current UK point of view.

DR. PILMANIS: Unfortunately we don't have the US Navy representative here. Would anybody want to venture any comment about the US Navy experience?

MS. MCGARVEY: I've always been told that the Navy's main concern was breathing following water entry. They decided that they would use 100% oxygen supplied through a demand regulator.

PROF. ERNSTING: Perhaps I can add more to the story. At the time we were designing their systems for four or five airplanes. You're absolutely right, they had lost quite a few aircrews sliding off carriers. Much of that was due to the design of their parachute harness release systems, but there were also huge advantages in having 200 liters of oxygen strapped to the back side and a regulator right at the mask so that they could breathe 100% oxygen.

The other thing that strongly influenced the aeromedical community in the USN was that during the same time period they had lots of toxic fumes incidents in their cabins, and pilots were quite disturbed. They hadn't conducted suitable evaluations of the panel mounted regulators and masks. If you're in the dilution mode or even if you're in the 100% mode in these pressure demand regulators, your mask cavity pressures can drop very quickly below ambient and contaminants can leak into the mask. I had long discussions with Captain Gale who was the chap responsible for the philosophy of going to 100% in the Navy and putting a regulator on the mask.

DR. PILMANIS: I suppose the question that is difficult to answer is, has it affected their operational performance?

DR. STOLP: I think the mechanism for the cough is certainly not the atelectasis. What is the mechanism of the cough?

PROF. ERNSTING: The thing that incites the cough is deep inspiration.

DR. STOLP: It probably has nothing to do with the atelectasis. Is it the pressure breathing?

PROF. ERNSTING: It has nothing to do with positive pressure breathing. This is ordinarily flying with 100% oxygen during high G. In all the very formal trials, and a lot of that data was from the Farnborough flight trials conducted in the 1950s on lung collapse, the pilots were doing anti-G straining maneuvers in flight on 100% oxygen and still got gross atelectasis that was found on X-ray after the flight.

F-22 Life-Support System for Altitude Protection

Dawn McGarvey-Buchwalder

Introduction

The F-22 Life-Support System is composed of both aircraft and man-mounted hardware. Both of these aspects are critical to the examination of the altitude protection afforded by this system. The aircraft mounted hardware is the product of the USAF's Tactical Life-Support System, B-1, B-2 and YF-22 programs. The man-mounted garments are based off of the knowledge and work of the USAF's Tactical Life-Support System, Combat Edge, ATAGS and the YF-22 programs. A system layout can be seen in Figure 1.
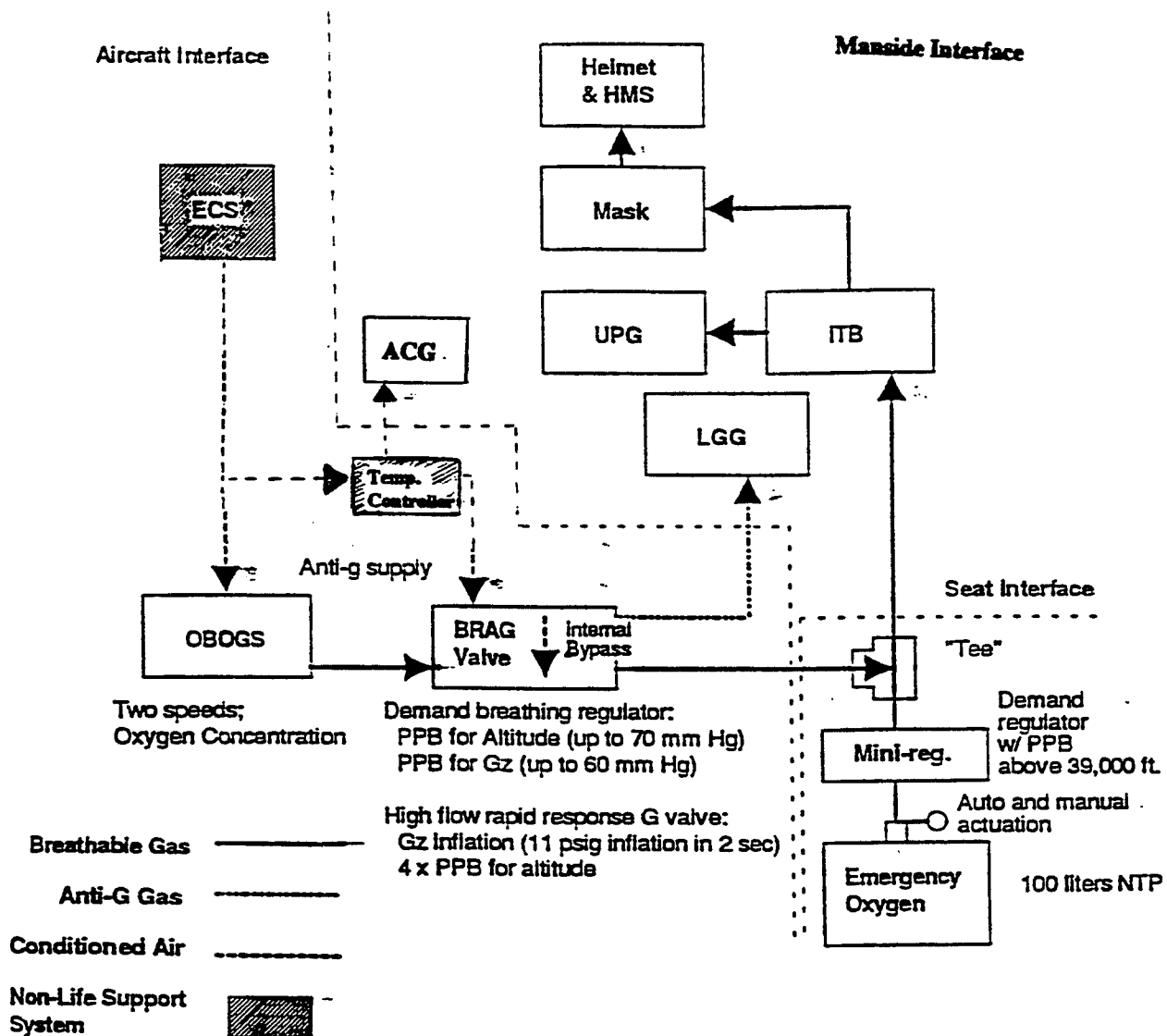

Figure 1. F-22 Life-Support System Interfacing.

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The F-22 program did not set out to create a system to expand the fighter communities altitude environment, but rather utilize the inherent altitude protection capabilities afforded by the partial-pressure garments for G protection. The system offers protection for in flight decompressions and post ejection to altitudes in excess of 50,000 ft. Due to the limited data base on partial-pressure systems with Onboard Oxygen Generating Systems, the altitude protection afforded by the partial-pressure ensemble is viewed as get-me-down protection.

Aircraft Mounted Equipment

Aircraft mounted equipment is composed of the Onboard Oxygen Generating System (OBOGS), Breathing Regulator and Anti-G (BRAG) valve and the Seat Mounted Emergency Oxygen System. Requirements of the breathing system include meeting the physiological requirements for all F-22 altitudes, acceleration level and emergency conditions. Requirements from ASCC 61/22 and 61/59 were drawn upon to establish the baseline breathing requirements.

Onboard Oxygen Generating System (OBOGS) CRU-104/A

The OBOGS is a three bed molecular sieve oxygen generating system which uses pressure swing adsorption to provide oxygen enriched breathing gas. Each bed is immobilized to prevent physical degradation. The inlet air is supplied from the Environmental Control System (ECS) which provides a temperature, pressure and moisture conditioned source. Nominally, ECS temperature is 85-90° F and pressure is 77 psig. However, the OBOGS has an internal pressure reducing valve which decreases operational pressure to 30 psig, approximately equivalent to ECS pressure at idle descent. Particulate filters are located on both the inlet and outlet air from the OBOGS. The OBOGS is initiated within the cockpit from an On/Off switch on the BRAG Valve panel.

The OBOGS has a two speed cycle design which automatically controls the oxygen concentration relative to cabin altitude. Between 0 and 11K ft cabin altitude, the OBOGS attempts to maintain a mean oxygen concentration below 60%. It maintains this mean concentration by switching between the low- and high-speed cycles. Oxygen concentration is monitored by an internal zirconia oxygen monitor (ZOM). Above 11K ft cabin altitude, the OBOGS operates exclusively at high cycle. This drives the oxygen concentration to the maximum that can be obtained, 94%. The actual maximum delivered concentration is a function of aircraft and cockpit altitudes, breathing load, ECS air characteristics and OBOGS bay temperature and any of these can degrade the concentration. The OBOGS can be commanded into the high-cycle speed at any cabin altitude through the Mixture switch on the BRAG valve panel. The OBOGS required performance schedule is shown in Figure 2.

The health of the OBOGS and product gas are continuously monitored by the unit and a status is sent to the Integrated Vehicle Subsystem Controller (IVSC). The four signals which are monitored are oxygen concentration, outlet pressure, controller fault or a oxygen monitor fault. The faults are monitored by the IVSC to screen for false warnings before sending a caution message to the pilot. All legitimate faults trigger a solitary message to the crew member which reads "OBOGS FAIL". All OBOGS faults sent to the IVSC will be captured by the maintenance panel for review at post flight. The maintainer will be able to isolate the failure to the specific fault.

Breathing Regulator and Anti-G (BRAG) Valve CRU-109/A

The BRAG valve combines the functions of both breathing regulator and anti-G valve into one package. This reduces size and improves the interdependent features required to drive both breathing and lower G garment (LGG) air for acceleration and altitude performance. The BRAG valve receives inlet breathing air from the OBOGS product gas, and conditioned ECS air for the anti-G air to the LGG. The BRAG valve has two outlets. The breathing gas from the BRAG valve is routed to the pilot via the breathing hose/line to the Emergency Oxygen regulator T located on the seat. The lower G air is routed directly to the LGG.
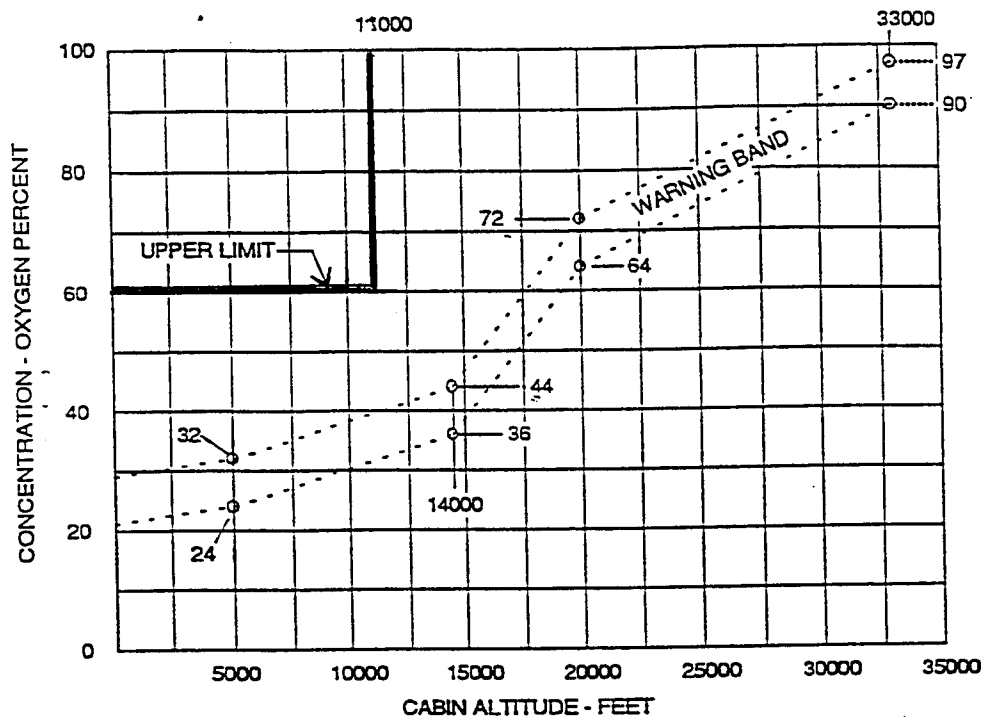
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Figure 2. OBOGS Required Performance Schedule.

The regulator portion of the valve is mechanical, delivering non-diluted OBOGS air on demand. The valve regulates the supply and pressure of the breathing gas to the pilot for both altitude and acceleration. The BRAG valve is designed for inspiratory demands to 210 liters per minute peak flow rate with low resistance. Additionally the BRAG valve provides a constant safety pressure of 1.0 to 2.0 inches of water gauge. Pressure breathing for altitude is automatically provided by sensing cabin altitude through an internal aneroid port. For altitude, pressure breathing is initiated at approximately 39,000 ft and reaches a maximum of 70 mm Hg at 53,000 ft, Figure 3. For acceleration, pressure breathing begins at 4 Gz with 12 mm Hg and increases to 60 mm Hg at 9 Gz, Figure 4. With acceleration pressurization, the BRAG valve requires initiation of LGG inflation before breathing pressurization will occur. This is necessary to prevent garment induced loss of consciousness by forcing blood away from the head.

The G-portion of the valve mechanically regulates the supply and pressure of air to the LGG for both altitude and acceleration. The anti-G system was designed to provide inflation of the LGG garment within 2 secs and the valve is integral to meeting this system requirement. For acceleration, pressurization of the LGG begins at 2 Gz and rises to a maximum of 11 psig at or above 9Gz, Figure 5. For altitude, the valve inflates the LGG to a 4 to 1 pressure as compared to the breathing air, Figure 6. [Note: This pressure ratio is currently being reduced; anticipated to be 3 to 1.] The BRAG valve does not require an operating OBOGS to inflate the LGG for acceleration protection. OBOGS product gas or emergency oxygen gas for pressure breathing for altitude is required for the BRAG valve to inflate the LGG for altitude protection.

The BRAG Valve is located on the right side of the cockpit. The valve panel is shown in Figure 7.

OBOGS ON/OFF Switch: It has an ON/OFF switch that powers the OBOGS. The OFF selection will close off the oxygen inlet in the BRAG valve. Switching the OBOGS to the OFF position will immediately send a caution to the pilot indicating OBOGS fail.
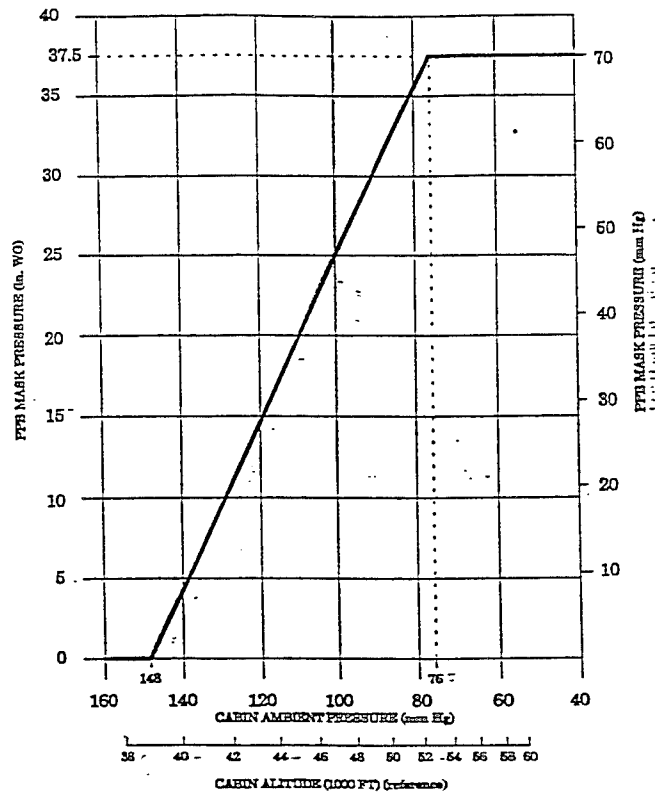
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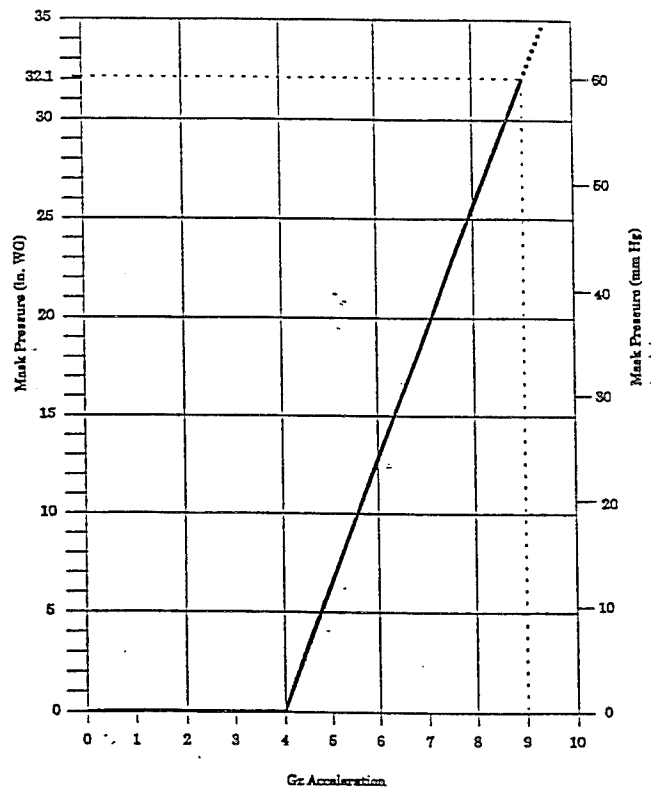**Figure 3. PPB vs Cabin Pressure Schedule.**

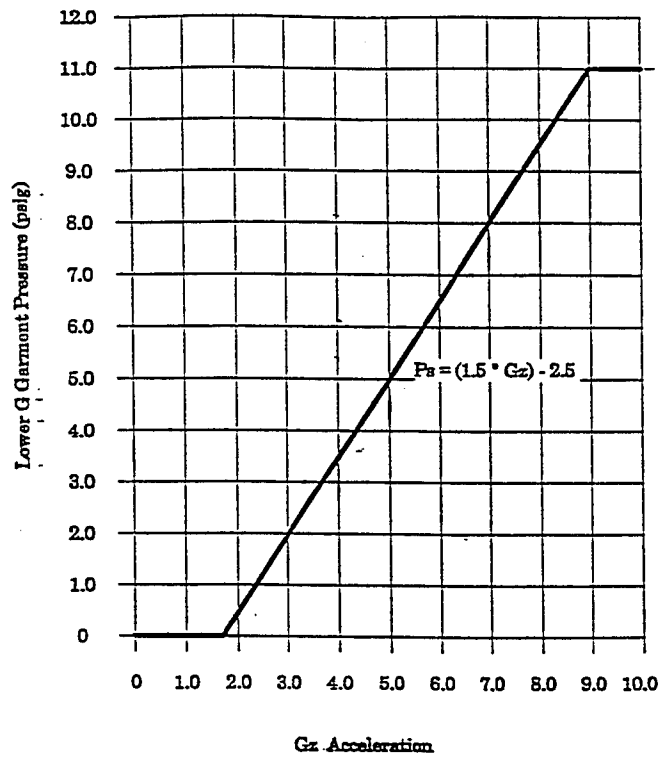**Figure 4. PPB Schedule vs Gz Acceleration.**

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Figure 5. Lower G Garment Pressure vs Gz Acceleration.
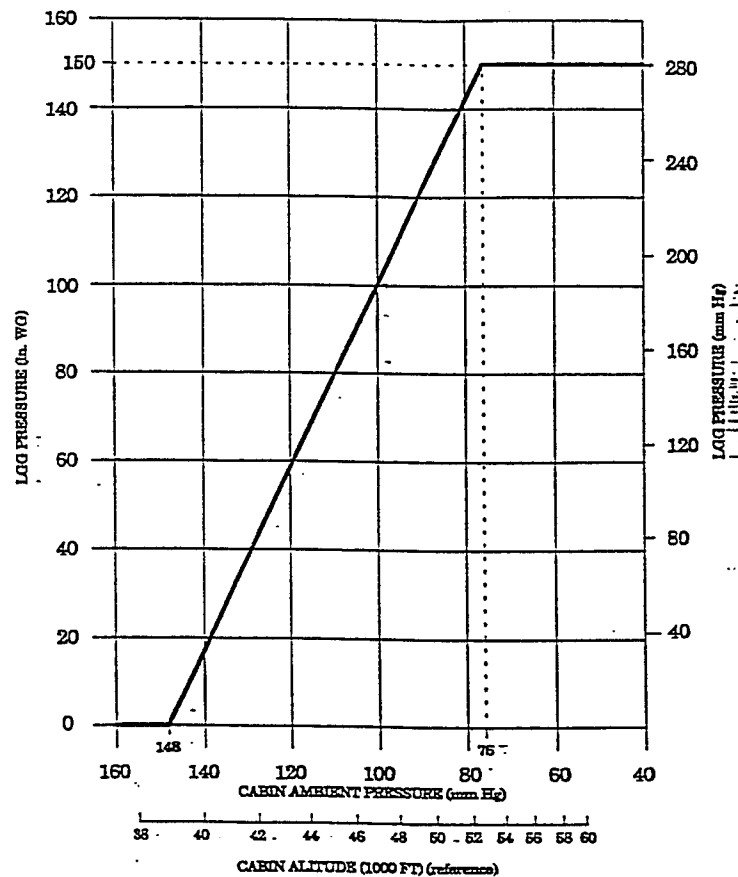

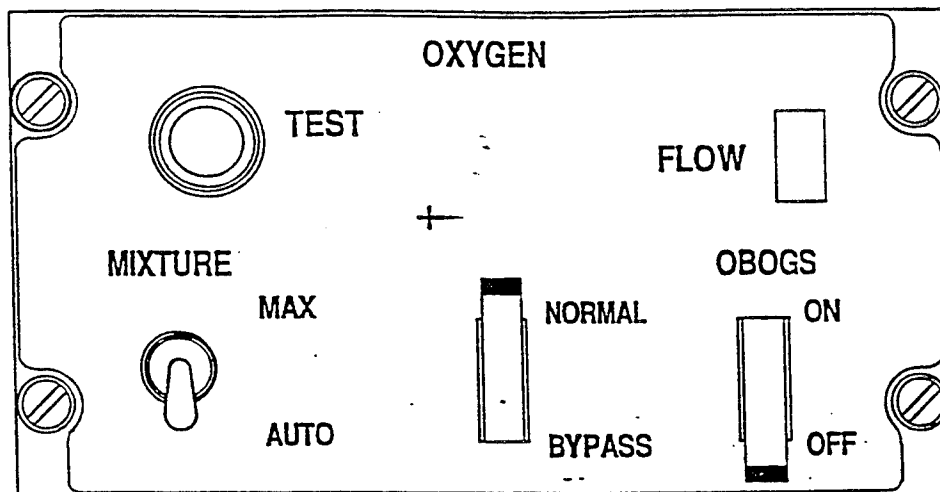Figure 6. Lower G Garment Pressure vs Cabin Pressure Schedule.

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Figure 7. Breathing Regulator and Anti-G (BRAG) Valve Panel.

NORMAL/BYPASS Switch: This switch was incorporated into the BRAG valve in the event of a loss of breathing gas from the OBOGS. The switch has two positions NORMAL and BYPASS. NORMAL setting provides breathing gas from the OBOGS. BYPASS setting channels air from the anti-G portion of the valve to the breathing side. This provides only 21% oxygen, but the air is filtered and conditioned by the ECS. The BYPASS setting would only be utilized in emergencies where the pilot wished to maintain the mask on and an increased concentration of oxygen was not required. The NORMAL/BYPASS and ON/OFF switches are interlocked to prevent inadvertent deactivation of the OBOGS.

Mixture Switch: The Mixture switch controls the cycle speed of the OBOGS. It has two positions NORMAL and MAX. NORMAL allows the OBOGS to automatically control the cycling according to the prescribed altitude schedule discussed in the OBOGS section. In the MAX setting, the OBOGS runs in high-cycle driving the oxygen concentration to the maximum amount that the OBOGS can produce.

Flow Blinker: The panel also has a pneumatic flow blinker which provides a visible indication of breathing gas flow. This information will enable the pilot to check for leaks, ensure flow and control breathing.

Test: This is a press to test feature that mechanically activates the anti-G portion of the G-valve, simulating a pressure breathing for G schedule.

Emergency Oxygen System

The emergency oxygen system is seat mounted and composed of a high-pressure oxygen bottle, oxygen hose and emergency oxygen regulator T. The emergency oxygen bottle is a 1500 psi, 50 cubic inch bottle, providing 100 liters of 100% oxygen located on the left side of the seat. The top of the bottle is mounted with a pressure reducer that decreases the pressure to 70 psi. The bottle is mechanically activated by a green ring.

Emergency oxygen is routed to the right side of the seat into the emergency oxygen regulator T. The regulator T serves several functions. It provides a location where the primary and emergency oxygen are tied into one system, shuts off the primary oxygen supply and provides pressure demand emergency oxygen at an altitude/pressure schedule. The regulator T uses a portion of the 70 psi emergency oxygen air to pneumatically shut down the primary (OBOGS) supply. The emergency oxygen supply then moves through the regulator and into the single output oxygen hose. When the emergency oxygen system is depleted, the primary oxygen is automatically

reinitiated. The benefits of this regulator T are that it minimizes the hose length required to obtain emergency oxygen, prevents dilution or contamination of the emergency oxygen supply, prevents dumping of emergency oxygen through the regulator as seen in current systems and provides another relief valve in the system to vent trapped gases on decompression.

A single oxygen hose is then routed to the Integrated Terminal Block (ITB). A quick disconnect exists between the BRAG valve and the emergency oxygen regulator T; this provides for seat aircraft separation.

Man-Mounted Equipment

The following equipment are utilized for standard F-22 flights and consists of the ITB, Upper Pressure Garment (UPG), air cooling garment, LGG, helmet and mask. All of the F-22 garments were designed to accommodate the central 99% of the USAF flying population. Requirements from ASCC 61/21 and 61/22 were drawn upon to establish the baseline breathing requirements.

Integrated Terminal Block (ITB) CRU-94/P

The F-22 will use the COMBAT EDGE CRU-94 ITB. The ITB distributes breathing gas from the BRAG valve to the UPG and mask. The ITB is mounted to the crew member's torso harness on the right side. If the UPG is not connected, the ITB pressure relief valve is designed to open at 35 mm Hg. This relief valve prevents exposure to increased breathing pressures. The emergency oxygen connection on the ITB is not utilized on the F-22 because it is not adequately sized for high-altitude protection requirements. As described in the emergency oxygen paragraph, the primary breathing system and emergency are tied together on the seat through the emergency oxygen regulator T. A pull apart disconnect exists between the T and the ITB; this provides for man/seat separation and connection/disconnection on ingress/egress.

Upper Pressure Garment (UPG) CSU-18/P

The UPG provides chest counterpressure to the pilot's upper torso at the same pressure as the mask. The UPG and the mask utilize the same breathing air from the BRAG valve, routed through the ITB. The garment is baselined off of the COMBAT EDGE upper pressure garment, CSU-17/P.

The garment is front opening, composed of a bladder that covers the chest extending over the shoulders and down the center of the back. The bladder is then surrounded by an exterior Nomex fabric. The UPG inflation port is located on the upper torso just right of the centerline. This is connected to the ITB. The fill/dump valve for the UPG is in the UPG connector at the ITB. The UPG is worn over the standard flight coveralls. It has a long shirt tail that can be tucked under the Lower G Garment to prevent the UPG from riding up during inflation. The UPG has a pass through on the left side for the air cooling hose to be connected to the air cooling garment when worn.

Lower G Garment (LGG) CSU-19/P

The LGG is a full-coverage G garment baselined off of the Advanced Technology Anti-G Suit (ATAGS) program. The garment provides pressure across the lower torso and legs to counteract the tendency for blood to pool in the lower part of the body during high-G maneuvers or during pressure breathing for altitude. The LGG is worn over the flight suit.

The garment is composed of a left-side and inner-leg zippers. Secondary connectors are located under the zipper to maintain garment positioning in case of a primary zipper failure. The internal single bladder provides complete coverage across the abdomen, and legs including knees and ankles. The bladder is then covered with an exterior fabric of nomex. The LGG inflation hose is located on the right upper hip. The LGG hoses (aircraft and manside) and connector have increased in size to maximize air flow and minimize resistance, assisting in achieving a G-system performance of suit inflation within 2 seconds.

Helmet -HGU-86/P

The HGU-86/P was specifically designed for retention during ejections to 600 KEAS, minimizing lift forces, providing maximum noise attenuation capabilities and accommodation of the central 99% of the flying population. The helmet is light weight and low profile, with a wide field of view and increased stability. It incorporates an earcup tensioning system which allows the earcups to be drawn into the head increasing the passive attenuation seal. The helmet will allow for the incorporation of the PRU-57 Active Noise Reduction System. The helmet is designed in conjunction with the mask, and therefore includes flexible receiver positioning to assist in mask integration/orientation. The features of this helmet are particularly beneficial in reducing physical fatigue.

Mask- MBU-22/P

The oxygen mask is designed for positive pressure breathing. The MBU-22 incorporates an automatic tensioning bladder on the front of the mask. This pushes the mask into the face versus alternatives where the face is pushed into the mask through a helmet mounted occipital bladder. The mask incorporates separate inhalation and exhalation valves to reduce breathing resistance and fatigue. It incorporates a reactive seal, and in conjunction with the automatic tensioning bladder, assists in holding pressures up to 70 mm Hg. The mask has a low profile and excellent visibility. The mask utilizes the AF standard M-169/AIC microphone, but it is positioned for improved communications.

Air Cooling Garment (ACG) CMU-31/P

The ACG distributes conditioned ECS air across the crew member's upper torso through a ventilation garment. The vest type garment distributes air through an open cell foam and is covered with a flame retardant material. The foam is lightweight and extremely pliable, providing maximum mobility. The ACG hose is routed on the left side of the garment and worn under the flight suit.

The air cooling system can control both flow and temperature. The maximum flow is 15 cfm and can be toggled to no flow through a flow restrictor on the aircraft side air cooling line. The temperature can be controlled through a rotary switch on the panel from 55 to 90 degrees F. The ACG is considered to be a critical provider of thermal relief from the additional heat stresses incurred with the external partial-pressure garments.

Additional F-22 Cockpit Information

The Environmental Control System is utilized to pressurize the cockpit to a differential of 5 psi from aircraft altitude. Cockpit pressurization is monitored by the IVSC and two faults could present a caution to the pilot. When cockpit pressurization exceeds the 5 psid schedule or cabin pressure exceeds 25,000 ft, then a "loss of pressurization" caution is provided. The first fault indicates slow leaks or a bad canopy seal, at low enough altitudes that the pilot should not have any high-altitude exposure. The second fault signals catastrophic pressurization failures.

Discussion

DR. SEARS: If you happen to be pulling G and you experience a rapid decompression during the high-G exposure at 40,000 to 50,000 feet, how does the system respond?

MS. MCGARVEY: It will give you the maximum pressure breathing schedule. The concentrator will probably still be functioning so you could continue to breathe OBOGS product.

CAPT. SCOGGINS: You indicated that you will probably conduct man rating in June 1996. Can you give us a better idea of your schedule beyond that in relationship to the aircraft flight test?

MS. MCGARVEY: Right now the IOT&E on the first vehicle is scheduled for June of '97. We have seven planes throughout that timeframe with production out to 2004. So between 1997 and 2004, we'll be doing several years of flight testing.

PROF. ERNSTING: Do you have an automatic emergency oxygen system. Is it controlled by PO2?

MS. MCGARVEY: No, the only time it functions automatically is during an ejection.

PROF. ERNSTING: Ejection, otherwise it's manual selection?

MS. MCGARVEY: That's correct. We did look at automatic activation of the emergency oxygen. Our reliability and safety people indicated that it was an unacceptable reliability-maintainability risk to use automatic activation of the emergency system.

DR. HARDING: Coming back to the question that Dr. Sears raised about experiencing a decompression while you are pulling G. Did I pick up from your diagram that your only pressure relief points are actually in the hardware? You've got nothing downstream in the garments for dumping gas following the rapid decompression during PPG?

MS. MCGARVEY: There's a dump valve at the chest mounted integrated terminal block. There is also a dump in the G valve line.

Eurofighter 2000 Life-Support System

A J F Macmillan, RAF, BSC, MB, ChB, MFOM

Introduction

Eurofighter 2000 (EF2000) will be the next generation interceptor introduced for service in the Royal Air Force. The aircraft has been developed jointly by United Kingdom, Germany, Italy and Spain. The consortium of companies comprising the development organisation has adopted the name Eurofighter. The aircraft is essentially lightweight, with a high power/weight ratio. Operational requirements for the aircraft include air combat capability, high sortie rate generation, ability to operate autonomously and have air-to-air refueling. It is anticipated that aircraft will have a service life of some 25 years. All aircraft systems and equipment are required to be user-friendly, thus simplifying operations, servicing and repair. Seven development aircraft are being built, two of which will be twin-seat. As in most modern aircraft, there will be extensive utilisation of multi-functional displays providing both systems information and operational data. Although the aircraft will be "fly-by-wire", the controls will have conventional positions with a central control column and left sided console throttles. The position of the control column is such that pilots' forearms and hands are positioned well below heart level, thus pilots may be susceptible to arm pain during positive pressure breathing for G protection.

Aircrew Equipment Assembly (AEA) and Life-Support System

Unlike previous UK aircraft, the aircrew equipment and life-support systems are defined as part of the weapons systems. Early in the development programme it was agreed that the four nations would adopt common equipment for acceleration and altitude protection, immersion and thermal protection, the head equipment assembly, NBC protection and associated other garments and supplies (e.g.,. liquid condition garments and personal equipment connectors).

Since the equipment is specified in the weapons systems, it is the responsibility of Eurofighter to develop and provide all of the AEA. This is a departure from the procedure normally adopted within the UK, and not surprisingly is giving rise to some difficulties particularly where complex equipment designs, for example the helmet system, are being evolved through the cooperation of specialist companies within a consortium. Consequently, the selection criteria for suppliers of component parts has not necessarily always related to technical merit, and compromises other than those required between physiological protection and operational suitability are more apparent in this programme.

Head Equipment Assembly

The head equipment assembly being developed for EF2000 comprises all the head mounted equipment. The helmet system is required to meet all the physical, physiological and operational needs of the four Air Forces to whom the aircraft will be supplied and is specified as part of the weapons system. The main requirements are summarised in Table 1.

Table 1. Head Equipment Assembly.

HEAD EQUIPMENT ASSEMBLY

INCLUDES ALL HEAD MOUNTED EQUIPMENT:

- Helmet System

- Mask/Respirator

- Liquid Conditioning Optional

REQUIREMENTS

Light Weight   not exceeding 1.9 Kg

Dual Visors

Comms with Active Noise Reduction (ANR)

Mask suspension

Enhanced Mask Sealing (EMS)

Helmet Mounted Display (HMD) and Night Vision Device (NVD) Integration

COMPONENTS:

Head/Helmet Position Sensing System

HMD - Inc various Cues - SAFE EJECTION

NVD - SAFE EJECTION

(Nuclear Flash Protection)

Laser Protection

Glare/Blast/Impact Protection

Oxygen Mask - NBC Respirator ('Automatic' Sealing)

Elec Power/Signal Connections

Possible Liquid Head Cooling

Life Support - Altitude Protection

The main components of the altitude protection system comprise a molecular sieve oxygen concentrator with a back-up auxiliary gaseous oxygen cylinder containing 100% oxygen. A seat mounted aircrew services. package (ASP) which provides the breathing regulator and anti-G valve supplies, and a partial-pressure clothing assembly. The aircraft cabin pressurization system is similar to that used in the Phantom aircraft and develops a 5 psi maximum differential pressure. A partial-pressure assembly comprising an oronasal breathing mask with enhanced mask seal (EMS), chest counterpressure garment which is integrated with the flotation garment (flight jacket), and full-coverage anti-G trousers (FCAGTS). The demand pressure breathing regulator is designed to provide 70 mm Hg pressure breathing at 60,000 feet. Cardiovascular support is enhanced by inflation of the anti-G trousers to a pressure of twice the breathing pressure at altitudes greater than 40,000 feet. Supply to the chest counterpressure garment incorporates a compensated dump valve so that pressure in the garment may be relieved in the event of sudden loss of cabin pressure when the garment is already inflated for its role in enhancing G protection. The components of the system in early development aircraft and those in the final system are summarised in Tables 2 & 3.

Table 2. EF 2000 Life-Support System--Interim Components.

EF 2000 LIFE-SUPPORT SYSTEM	
INTERIM COMPONENTS	
MSOC -	PO₂ CONTROL BY PO₂ SENSORS
ANTI-G VALVE -	HAWK
DEMAND REGULATOR-	HARRIER GR7 (TYPE 600 WITH PBG) TORNADO PEC
AEA -	PRESSURE WAISTCOAT
	FCAGTS
	INTERIM HEAD EQUIPMENT ASSEMBLY (HEA)
	NATIONAL ALPHA TYPE 700 HELMET
	TYPE P/Q MASK WITH ENHANCED SEALING
	NATIONAL/UK GFE (UNDERWEAR ETC)
SEAT -	TORNADO MK 10LE

Table 3. EF 2000 Life-Support System--Final System Components.

<u>EF 2000 LIFE-SUPPORT SYSTEM</u>

<u>FINAL SYSTEM COMPONENTS</u>

MSOC -	CONTROLLED FROM CENTRAL COMPUTER
ANTI-G VALVE -	)
	) AIRCREW SERVICES PACKAGE
DEMAND REGULATOR -	)
AEA -	FLIGHT JACKET
	FCAGTS
	LIGHT WEIGHT COVERALL
	THERMAL PROTECTION
	IMMERSION PROTECTION
	INTERIM HEA INITIALLY
	NATIONAL GFE
AT A LATER DATE	FINAL HEA (Avionics Helmet)
	PERSONAL LIQUID CONDITIONING
	NBC PROTECTION

Life Support - G Protection

EF2000 will be capable of maintaining accelerations of up to +9G$_z$ at high rates of onset. The main components of the G protection system comprise the anti-G valve, pressure demand breathing regulator, full-coverage anti-G trousers, chest counterpressure garment (flight jacket) and pressure breathing oronasal mask with enhanced mask sealing. The high flow capacity anti-G valve utilises a standard pressure schedule and has a flow capacity which requires the full-coverage anti-G trousers to reach 90% of final pressure within 1 second of G onset. In common with most other advanced breathing systems for G protection, a signal from the outlet of the G valve initiates the pressure breathing within the demand regulator. At the present time, pressure breathing cuts in at 2G with delivery pressure rising linearly to 60 mm Hg. The current programme for development flying is shown in Table 4 with the various combinations of equipment to be utilised in the programme. The final life-support equipment incorporating all components of the system is planned to be installed late in 1997/early 1998. Retrospective fitting initially to aircraft DA/06 will be carried out first.

Table 4. EF2000--Crew Systems Programme.

	DA 01/02/03	DA 04/05/06/07	DA 01/02/03	DA 06
MSOC	Interim	Interim	Final	Final
AGV	Hawk	ASP	ASP	ASP
Regulator	Type 600+PBG	ASP	ASP	ASP
AEA	Interim	Final	As DA 04/07	As DA 04/07
	CCPG	Flight Jacket		+NBC
		Coverall Lightweight		+LCG
	FCAGTs	FCAGTs		+HEA
		TPG/IPG		
	HEA Interim	HEA Interim		
	EMS Mask	EMS Mask		
	UK GFE	National GFE		
Seat	Mk10LE	Mk16A	Mk16A	MK16A

Table 5. Eurofighter 2000 Life-Support System--Some Physiological Challenges.

EUROFIGHTER 2000 LIFE-SUPPORT SYSTEM

SOME PHYSIOLOGICAL CHALLENGES

ACCELERATION PROTECTION

    OPTIMISE PBG SCHEDULE

    CUT-IN

  G THRESHOLD

      SLOPE OF PBG SCHEDULE

      MAXIMUM PBG

    MINIMISE ARM PAIN

      COCKPIT CHANGES

      PBG SCHEDULE

    LOSS OF CABIN PRESSURE DURING PBG

ALTITUDE PROTECTION

    OPTIMISE PRESSURE RATIO BETWEEN ANTI-G TROUSERS AND MASK

    ESTABLISH ACCEPTABLE DEGREE OF HYPOXIA

      MSOC GAS COMPOSITION PRIOR TO DECOMPRESSION

      CONFIRM PBA OF 80 MM HG ACCEPTABLE

      EFFECTS OF TRAPPED GAS IN NBC ASSEMBLY

Conclusion

EF2000 life-support system will incorporate state of the art anti-G breathing supplies with Aircrew Equipment Assemblies comprising full-coverage anti-G trousers, chest counterpressure garment and pressure demand oronasal mask with enhanced mask sealing. Final design standards for much of the equipment to be used in service e.g.,. helmet equipment assembly and NBC protection, have yet to be established since there are still a number of physiological challenges to be solved (Table 5). However, there are sufficiently mature life-support equipment already developed which, if necessary, may be utilised so that the development programme for the airframe itself is not compromised.

Discussion

DR. MACMILLAN: A few additional comments on the life-support systems. The final molecular sieve oxygen system, in the Eurofighter 2000, will be controlled from the central computer and will respond and be controlled by many more variables than just the PO2 that's delivered. Such things as inlet pressures, outlet pressures, humidity, cabin altitude, aircraft altitude, will all be taken into account in controlling the mixture available from molecular sieve. The auxiliary 200 liter oxygen system in the Eurofighter will be activated automatically in the event of concentrator failure, cabin altitudes exceeding 25,000 feet, (because our molecular sieve will be giving less than 70% oxygen mixture) and also come on automatically on ejection.

The various Air Staff's have also asked us to relook at the concept of operating this aircraft in a chemical environment. It's believed that the threat of chemical attack has been reduced, but with the proliferation of agents, it is very unpredictable on how they will be used. In a similar fashion to wearing full-pressure suits, the aircrew wouldn't like to have their head in a bag and operate that way for NBC protection. So it's the same sort of encumbrance, lack of mobility, visual fields and so on, that has caused them to ask the life-support community, do we really need chemical protection? Is it possible to keep the cockpit clean considering the reduced threat? If it isn't then we will be required to have a backup respirator and individual protection system.

DR. SEARS: Why did you have a question mark by liquid conditioning for thermal comfort?

DR. MACMILLAN: It was against the headgear. That's one of the physiological challenges that we still need to resolve. With adequate body cooling do you need head cooling?

MAJ. MILLER: How are you handling low oxygen spurious warnings on your system?

DR. MACMILLAN: Are you talking about the temporary drop outs?

MAJ. MILLER: Yes.

DR. MACMILLAN: That will be addressed in the final system by a software package.

HIGH-ALTITUDE RECONNAISSANCE OPERATIONS

Wilbur T. Workman, Colonel, USAF, BSC
Edward A. Walby, LtCol, USAF

Introduction

As I travel around, I am often asked what I do in the Air Force. When I tell them I am responsible for providing physiological support for the USAF High-altitude Reconnaissance Program, with its mainstay--the venerable U-2 aircraft, I am continually amazed at people's reactions. Many are surprised the U-2 is still flying. It is the purpose of this paper to briefly describe the U-2 program and to focus principally on current physiological support requirements that directly impact high-altitude reconnaissance operations. The operational requirements for full-pressure suit use, its limitations, integration problems, issues of wear, and the potential for use in high-performance aircraft will be discussed.

Background

On that fateful first day of May 1960, the world was openly introduced to the phenomenal U-2 when Francis Gary Powers was shot down over the Soviet Union. Even though there was limited knowledge of its existence prior to that date, this event propelled the U-2 into international prominence sparking a Cold War crisis.

Designed in the early 1950s, the first U-2 flight was on 6 August 1955. Having undergone slight modifications over the years, the current U-2 is a high-altitude, high-aspect ratio aircraft that is relatively light weight and capable of flight in excess of 65,000 feet. Its construction is simple with pulleys, cables, stick and rudder. The cabin is pressurized to 30,000 feet while flying in the tropopause free of clouds and most wind. The U-2 "R Model" can fly in excess of 11 hours while the newly re-engined "S Model" can fly longer than 16 hours without external pods. Neither model is capable of mid-air refueling. The basic design with bicycle landing gear was made to minimize drag and weight and causes unique handling characteristics. This design was made to minimize drag and weight. On landing, the airplane must be brought to a complete stall at 1' to 2' off the ground prior to touch down. Peripheral vision is extremely critical during this phase. Due to the limitations imposed from the full-pressure suit, a mobile (extra pilot in a chase vehicle) is used to assist in landing. The mobile pilot also serves as a backup pilot for high-priority missions should the primary pilot become incapacitated prior to flight.

The U-2 is the premier high-altitude manned reconnaissance collection system capable of collecting IMINT (imagery both radar and literal) and SIGINT or signals intelligence. The system consists of aircraft, sensors, data links, and ground processing facilities. There are 27 combat coded aircraft with three designated backups. There are four 2-seat trainers and four aircraft at Palmdale Flight Test. One aircraft is on loan to NASA. The sensors consist of an array of Advanced Synthetic Aperture Radar (ASARS) that provides near literal imagery; electro-optical sensors that provide essentially "television like" still pictures; wet film cameras that use conventional film requiring development; and, other classified sensors. Data links are maintained by either line of sight or satellite coverage. An integral part of the manned reconnaissance system are the ground processing facilities. The Mobile Intelligence Processing Facility or MIPE develops wet film. Only one MIPE exists. Electronically derived intelligence is processed by the Contingency Airborne Reconnaissance System (CARS) of which only two exist. These processing systems can also connect with some Army and Navy ground stations. Peacetime taskings come directly from the National Command Authorities and are approved by the Joint Chiefs of Staff. Wartime taskings come directly from the theater commander.

The body's physiological response to extreme altitudes is well documented and has been discussed extensively during this workshop. It is not my intent to cover that information again. However, a short review of the current S1031 full-pressure suit is in order. The suit used by U-2 pilots is considered a backup system for survival at altitudes in excess of 50,000. In other words, it is considered a backup cabin pressurization system. The suit only inflates as the cabin altitude rises above 35,000 feet. In normal flight, the suit is unpressurized and provides 100% oxygen in the helmet's breathing compartment. Ventilation is provided by circulating engine bleed air through the suit. Should cabin pressurization be lost at altitudes greater than 35,000 feet, the expanded air is trapped in the suit by the suit controller to maintain the pressure in the suit equivalent to 35,000 feet or 3.5 pounds per square inch (psi).

The 3.5 psi level was chosen as a compromise between protection and mobility. If pressurized greater than 3.5 psi, it becomes extremely rigid and impedes mobility. The principal difficulties are moving the arms and closing the gloved hand. If pressures less than 3.5 psi were used, mobility would be increased but oxygen would have to be provided to the helmet under increased pressure requiring the pilot to pressure breathe. Additionally, the risk of decompression sickness (DCS) would increase. With normal cabin altitudes above 25,000 feet, additional protection is required to minimize the risk of DCS. This is accomplished by prebreathing 100% oxygen for at least one hour prior to takeoff for all missions above FL450 (45,000 feet).

Routine High-Altitude Reconnaissance
Mission Support Operations

The 9th Reconnaissance Wing, Beale AFB CA is responsible for the worldwide employment of the U-2 high-altitude manned reconnaissance system. From its home station at Beale, the system is deployed to four operational locations throughout the world. Contingency deployments are conducted as needed. The number of aircraft, personnel, and sortie rate at a given location are determined by the operational need. From a physiological support perspective, this requires an infrastructure of over 140 highly trained technicians and currently requires approximately 100 temporary duty (TDY) days per year per technician. This technician complement is made of an Air Force unique mixture of dedicated aerospace physiology and life-support personnel. The full array of life-support equipment: full-pressure suits, helmets, seat kits, survival kits, parachutes, torso harnesses, etc. are maintained by 9th Training Squadron personnel (better known as PSD). In order to maintain mission responsiveness and quality control, all suit and oxygen regulator overhauls and suit maintenance activities are conducted by squadron personnel. The only exception to this are major factory suit overhauls which are conducted every six years. Without this local depot level function, the 9th Wing mission would often be compromised.

In many ways, the preflight routine for the U-2 pilot is similar to that of other weapon systems. That is, preflight crew rest standards are observed. However, there are other restrictions that he or she must adhere to. For example, the pilot must observe a very conservative dietary intake the day prior to a scheduled flight and attempt to maintain a regular bowel cycle. The need to regulate bowel movement is vital to the success of any lengthy mission since bowel movements during flight cannot be accommodated. Urine management is effective; feces management is impossible.

Showtime is 2hr/45 minutes prior to launch. A meal with the mobile officer is usually the first event. The mission pilot can choose a meal from a high-protein, low-residue menu to maximize energy levels and minimize the need for a bowel movement in flight. The flight plan is reviewed at Base Operations if it is a stateside mission. If deployed to a forward location, an intelligence and mission overview briefing is provided instead. Once this is completed (usually about 1+45 prior to launch), the pilot attempts a bowel movement. Preparation for suit-up begins about 1+30 prior to launch. This ritual includes an abbreviated physical to check for blood pressure, temperature, and ability to equalize pressure in the middle ear and sinuses. Equally important is an eye to eye assessment with the pilot by the support technician. The importance of this last minute hands on evaluation before the pilot is "buttoned up" so to speak cannot be overstated. Actual suitup begins 1+15 prior to launch.

Prior to a scheduled flight, the mission pilot's suit is inspected and then laid out for pilot/suit integration. A team of three highly trained technicians are responsible for the dress procedure: two actually dress the pilot and one safety supervisor oversees the entire operation. Once integrated, a thorough systems check is conducted to ensure proper suit operation. At this time, the pilot begins the one hour denitrogenation period. While the pilot is being suited up, the mobile pilot conducts a preflight on the jet. Once completed, the mobile informs support personnel that the jet is ready for the pilot. The pilot is then connected to a portable ventilator to provide suit ventilation and 100% oxygen to maintain an uninterrupted denitrogenation cycle and escorted to a waiting van for transportation to the aircraft. The same team stays with the pilot and is responsible for integration into the aircraft. Prior to removing the pilot from the van, technicians inspect all life-support equipment components for proper installation. Once complete, the pilot is assisted into the cockpit where physiological support technicians ensure that all suit connections to the aircraft life-support and intercom systems are properly made. This team remains on the flight line until the pilot has been successfully launched. At the end of the sortie, the entire procedure is reversed. At the completion of each high flight, the pressure suit is inspected for any discrepancies that resulted from operational use. This ensures the suit is ready for subsequent use.

Full-Pressure Suit Limitations

There are numerous physiological, psychological and physical limitations associated with the wear of a full-pressure suit. The physiological limits range from the inability to clear trapped air spaces at higher altitudes to extreme thermal stress. As previously stated, the importance of the preflight physical cannot be emphasized enough. Unlike routine fighter operations where the pilot can readily perform the valsalva maneuver, a U-2 pilot cannot open the helmet visor at higher altitudes for oro-nasal access. Flying with a minor upper respiratory infection can prove disastrous for the pilot. Suit ventilation is provided by engine bleed air. During ground operations, high cockpit temperatures combined with low suit ventilation rates can be devastating. Even if a rise in core temperature can be avoided, hydration levels can be severely affected. Dehydration combined with elevated suit altitudes up to 35,000 feet increases the risk to DCS. Because of the added weight and bulk of the suit, pilot fatigue is critical on long missions. Most U-2 pilots remain as motionless as possible attempting to maximize energy conservation for the most demanding phase of flight -- the landing. This is significant in that the pilot is faced with the highest task saturation and physical demand when he or she is most fatigued. The eyes become dry and blood shot due to elevated face heat and the 100% oxygen environment in the helmet face compartment. Gastrointestinal health is a necessity. U-2 pilots are more attuned to a thorough knowledge of critical bodily functions than many other pilot types. Even a mild case of food poisoning can be deadly. One actual instance of poorly cooked chicken almost incapacitated a pilot before he could return. The awareness level for minor aches and pains is also heightened because of the increased rate of DCS in the U-2. Peripheral vision is reduced in the helmet and poses a major limitation on landing that is, once again, the most difficult phase of flight.

Psychological limitations are harder to define but no less important. There is a small percentage of prospective U-2 pilots who are unable to cope with the confines of the suit and become claustrophobic. In most cases, this is observed and documented prior to acceptance into the program. One of the principal aims of the pre-acceptance interview for the pilot is to determine suit compatibility. If the pilot cannot tolerate the suit, he or she is not accepted into the U-2 program. In fewer instances, a pilot develops an aversion to the suit after entering the program. In these cases, apprehension usually appears during the second long TDY. Perhaps this results from the pilot developing more confidence in flying ability that allows more time to focus on personal well-being. On one occasion, a pilot developed an ulcer while deployed and was returned home. The sound of breathing (or the Darth Vader Syndrome) is difficult for many to ignore, especially for the first few minutes in the suit. It is not uncommon for some degree of anxiety to result. For all pilots, there is a certain amount of mental "pumping up" before a long mission. For most U-2 pilots, this is more pronounced. However, most anxiety disappears as cockpit integration is complete and mission focus becomes primary.

If you consider pressure suit application to the high-performance fighter environment perhaps physical factors are the most limiting. At lower cabin altitudes, the suit feels soft or mushy. However, at higher cabin altitudes resulting from a loss of cabin pressurization, the suit is hard and rigid. In this instance, movement of the

extremities is from sheer physical strength and seriously impairs a pilot's reaction time. Manual dexterity is severely affected by glove inflation. Even in the "soft" state, tactility is reduced with the current glove design making buttons and switches harder to activate. Extreme head movements are not possible without physically assisting this action by grabbing the helmet's bailer bar with the hand. The added bulk of the suit, combined with cockpit confinement, often results in dropped items remaining on the floor during flight.

Potential for Use in High-Performance Fighters

The design of current operational full-pressure suits makes their use in high-performance fighters ill-conceived. From a tactical perspective, the ability to maintain optimum head movement to maintain a "heads up" posture is severely compromised. Pressure suit helmets in use today severely reduce peripheral vision. This, combined with the inability to conduct rapid head movements reduces the ability to conduct visual searches and detracts from the pilot's situational awareness. Current fighter cockpit designs are not capable of accommodating the ventilation requirements of current full-pressure suits. Thermal stress will be commonplace without cockpit modifications. Even in the unpressurized state, the added bulk and reduced mobility from the suit will negatively impact a fighter pilot's reactions. In the fully inflated state, the pilot's overall mobility will be seriously degraded.

From the operational support perspective, expanding pressure suit support to the current fighter community is equally difficult. The physiological support infrastructure required to maintain the operational readiness of the U-2 fleet is robust. Expanding this responsibility to a larger, more diverse complement of weapon systems cannot be accomplished with current resources. Wing level life-support shops have been providing superb support to the fighter community for years but are ill equipped to handle this new responsibility. Maintaining full-pressure suits is logistically intensive. A large infrastructure must be retained to conduct required inspections and repairs; to conduct suit donning and doffing procedures; to ensure proper pilot/aircraft integration; and, to provide for forward deployed support.

Conclusion

Given the current state of the art for full-pressure suit operations, today's high-performance fighter operations cannot be supported. To do so requires new, innovative suit design initiatives to produce a suit capable of self donning and doffing, one with integrated self diagnostics and disposable suit components, and reduced bulk especially over the extremities. The final suit configuration must be one capable of flawless, sustained performance without the current level of required resources and maintenance effort.

Discussion

COL WORKMAN: A few additional comments on the new S-1034 full-pressure suit that will fielded shortly. We have successfully met the requirement of reduced weight and reduced bulk, increased mobility, and also now have an improved articulated glove connection which allows better mobility of the hand and less arm fatigue. We've gone to the single layer Gortex, which was mentioned earlier. But the Gortex leaks like a sieve in terms of chemical protection. So we do not have chemical protection in this particular suit.

We've also incorporated a new lightweight helmet. The pilots have responded enthusiastically to both the helmet and suit prototypes. They did, however, want the same level of thermal protection that we had with the S-1031. I believe that pilot acceptability will increase even further with the addition of a newly developed thermal liner.

LT. COL. DEMITRY: What's the current status of the female urine collection system?

COL. WORKMAN: We're incorporating the male urine collection device (UCD) concept into the disposable absorption containment trunk (DACT) concept and it's working quite well. We've done some prototype testing on it with our young female aircrew and it really is promising.

LT. COL. DEMITRY: Does the DACT directly absorb the urine?

COL. WORKMAN: No, the DACT only absorbs what may leak from the system.

DR. WEBB: Do you attempt to control activity the day prior to the flight?

COL. WORKMAN: We encourage them to reduce strenuous activity.

COL. WORKMAN: One thing I also need to mention is the cost of the new S-34 full-pressure suit. The average cost of a the newer suit is about $96-98,000?

MR. ELLIOTT: Around $100,000.

COL. WORKMAN: By the time you issue the pilot two suits, helmet assembly, torso garment, you're looking at in excess of a quarter of a million dollars per pilot. Logistically, with the current state of pressure suit technology that we have, and with our current manning structure, it would be very, very difficult for us to maintain the logistical tail required to support that in a fighter type operation. Mr. Greg Elliott, the item manager of the suit is here; between he and I, we should be able to answer any questions that you might have regarding the suit.

DR. WEBB: Can you give us the pilot/aircraft ratio to give us an idea how much the suit cost is versus an upgrade of the pressurization system on the aircraft?

COL. WORKMAN: One of the things that has been established as a defined need is a cockpit upgrade. Increasing the cabin pressure schedule has been suggested, but consider that the U-2 design goal was to conserve weight. We've added a new engine that has increased the performance of the airplane by giving it additional range and altitude capabilities. A recent decision has been made to set aside the cockpit upgrade and fund defensive systems for the airplane. I don't see that priority changing, when you consider that the role of the U-2 is slowly changing.

MAJ. MILLER: Can you comment on the capacity of the liquid oxygen system and what type regulator you're using?

COL. WORKMAN: Col. Sherman, do you remember what the capacity of the LOX converter is on the airplane?

COL. SHERMAN: I think ten liters, but I'm not sure.

COL. WORKMAN: The cockpit equipment is fairly old technology. If you ever have an opportunity to look inside one, you'll be in awe how ancient the systems are.

MR. ELLIOTT: I'd like to add a comment regarding chemical defense protection. W. L. Gore does have a material that we are evaluating now for the bladder. It's known as Chempac and it's a trilaminate breathable material. We have tested it already with live agents and it worked very well. David Clark is looking at that now as a product improvement if the need arises. We don't have a current requirement for chemical protection in the suit.

DR. SEARS: The differential pressure schedule is still only 3.5 psi?

COL. WORKMAN: Yes, 3.5 psi.

DR. PILMANIS: As you know, pilots don't report DCS. If you look at laboratory results relevant to the profiles that U-2 pilots fly, the DCS risk runs anywhere from 60-70%. That frequency has not been reported operationally, but that is the risk. We did three surveys of retired and active U-2 pilots and the data confirmed that the risk is there. The common response to the DCS frequency is, "so what, we complete our mission without an impact". However, there is an operational impact of approximately 34%, as admitted by the U-2 pilots. About 16% of the pilots actually alter their mission profiles because of DCS. Around 13% of the pilots experienced neurological involvement. So there is a mission impact. These are surveys, not research type data, but nevertheless I think that the lesson is that cabin pressurization in aircraft should not stay at these low levels in future high-altitude aircraft. We didn't have the data in the 1950s, but we do have the data now and it should be used.

A Panel on
Sustained Operations at High Altitude

MODERATORS:

      MAJOR PETER DEMITRY , HSC/XRT, BROOKS AFB, TEXAS
      DR. KEN ACKLES, DCIEM, NORTH YORK ONTARIO, CANADA
      COL. RONALD HILL, AL/CF, WRIGHT PATTERSON AFB, OHIO

DR. PILMANIS: Let me define my original concept of the panel on sustained high-altitude operations. It has been generally agreed that a get-me-down scenario from 60,000 feet is achievable. It's probably not even a major issue. By sustained operations, what I had in mind was a matter of 5 or 10 minutes. Physiologically, going from 1 minute to 5 or 10 minutes is a large step. If it is operationally necessary, the research data must encompass all the issues following loss of pressure for these longer periods of time. My original idea was to assess the importance and issues involved in remaining at 60,000 feet for up to 10 minutes after loss of pressurization. I doubt that we need, at least in the US Air Force, to look at longer exposures.

LT. COL. DEMITRY: Sustained operations really depends on the aircraft mission. If you were to go up for 8 or 10 hours in the U-2, and then come back after being in a full-pressure suit, to a night time landing at a remote base with weather, this is probably one of the most challenging things I think an aviator can do. So as far as fatigue and all those other kinds of things that play into this scenario, it truly is a challenge to those guys. And having done it once, I have no pressing desire to ever do it again.

When a pilot thinks of the flight profile, he normally considers both the penetration and employment phases. Let's presume you want to go long distance. Normally you want to go as high and fast as you can until you start requiring afterburner or incur some other penalty such as friction drag and leading edge temperatures. To give you an idea of the velocity, Mach 1 is about 10 miles a minute. If I can stay at high altitude for a few minutes, I can put distance between a threat on the ground or a threat in the air. And if I'm going that fast his missile or whatever he's going to use is going to have a very difficult time reaching me, especially with a negative closing velocity or opening distance between airplanes. Major Neubeck discussed the concept of an engagement zone and I think his graphics were excellent, but you have shrinking cones and the faster you're going, you're going from a poor to an expert gunner in order to successfully target my aircraft. Major Neubeck brought up something that I had never really considered before. The kinetic energy of a missile at the higher altitude is superb, so whoever goes up to the high ground should have a real advantage. As far as reconnaissance goes, if the aircraft can go high, the logic would be compelling for going higher, staying out of the threat and having a standoff capability for a precision strike. I have no definite requirements in mind, but someday I'm sure they'll be there.

The concept of sustained operations between organizations, as all would likely agree, is very different. In high-performance aircraft, I do see the need for a 5 or 10 minute ability to work my way through a SAM belt if it's possible, but everything comes down to an issue of balance. What is it going to cost me in terms of performance, complication, maintainability, in order to get this additional capability?

Based on what I know about the needs and what I would be asking the research community, is that if I am at some nominal altitude, 70,000 feet for starters, and I lose pressurization with other than a full-pressure suit, tell me what I need to do in order to keep my airplane as high as possible with some acceptable risk. I want to remain as high as I can, possibly stepping down from 70,000 feet to 50,000 feet and stay there for some period of time and subsequently to some lower altitude for another period of time, so that the risk remains constant. Provide me with some sort of a schedule of the risk, because this is all that the pilot is going to be able to remember, kind of a rough rule of thumb for four or five altitudes and periods of time. That would allow him to get through a threat, keeping as high as possible. A scenario that comes to mind is that I'm well into enemy territory, and I want to get high over a SAM belt going Mach 2 (20 miles a minute). If you can provide me with 5 minutes, that's 100 miles. You're going to have to be a respectable gunner or you're going to have to be a fairly sophisticated threat to hurt me before I can get into friendly territory.

The second scenario is that I've got a long way to go to return home. I may be deep into the enemy's territory and have a concern regarding fuel consumption. I need to keep my airplane very high, because if I come down to lower altitudes my fuel consumption goes up. What is the highest altitude following decompression that I can fly for a nominal couple of hours? Why a couple of hours? It's a starting point.

MAJ. NEUBECK: We are discussing two different schedules. One where you maintain a certain altitude for a certain amount of time, maybe a minute or 2 minutes, then you work your way down, eventually getting down to a safe altitude. Another profile was to maintain the high altitude for as long as possible and then descend quickly to a much lower altitude. From a threat standpoint, the safest condition will probably be to maintain the higher altitude for a longer period of time.

LT. COL. DEMITRY: Let us select an altitude to be more concrete.

MAJ. NEUBECK: Let's use 55,000 or 60,000 feet.

LT. COL. DEMITRY: Assume you're at an operationally representative altitude around 60,000 feet. You have experienced a rapid decompression. There ought to be a rule of thumb, that the pilot is familiar with, that will allow him to maintain the highest altitude for the longest period of time. It's kind of like a reverse diving table, where you may have some intermediate flight levels with an acceptable risk of DCS or hypoxemia while maintaining the highest altitude for the longest period of time. Also, what is an altitude that I can descend to and stay at for a nominal two hours or so? Does that make sense as a research question?

COL. HILL: Every time you stop at one more level, you have to establish a confidence interval for the preceding level. Your confidence level becomes unacceptable after you make a few stops.

MAJ. CAULKINS: A question appropriate to the F-22 at least, is to determine the probable reaction of the pilot as regards the oxygen system with a loss of cabin pressurization at 60,000 feet? Will you get a OBOGS fail light and will the pilot need to use his emergency oxygen bottle?

MS. MCGARVEY: Is 93% oxygen going to be an adequate concentration at 60,000 feet?

COL. HILL: Before moving to specifics I think we should let Lt. Col. Demitry continue with his concept.

MAJ. CAULKINS: I think for the F-22, this might be the determining factor. Once the pilot activates his emergency oxygen (if he needs 100% oxygen or if the OBOGS gives him a fail light) his only alternative is to activate the emergency oxygen. If he does that, the emergency oxygen will shortly be gone and he will be unable to stay at these higher altitudes.

LT. COL. DEMITRY: Your logic is compelling and I don't for a second think that this approach is sacrosanct. What I'm hoping for is that I may stimulate a better methodology from this group with which to go looking for answers. Once we have an intelligent approach to the problem, then each specific system must be reviewed to establish their individual constraints.

COL. HILL: I have to congratulate Lt. Col. Demitry because I think he is proceeding in the right direction. Yesterday I asked the question what are we supposed to give the user? He is trying to phrase what our answer could possibly look like and not what the answer is.

LT. COL. DEMITRY: Clearly I would like a full-pressure suit that I, as fighter pilot, would feel totally unencumbered. My initial perceptions are that I would eventually like to get an automated system that can keep up wherever I want to take my vehicle. We have that in a lot of other systems and a lot of other applications. We have automated threat recognition which has already been demonstrated and a lot of my background and bias is from advanced fighter technologies out at Edwards AFB. We need the same thing in life support. The threat, instead of being a surface to air missile or another airplane, is the ambient environment; the hostile environment. We must

have some sort of model before we can sort out all the many variables. We still are pretty primitive with our true understanding of the modeling, but it is an approach that should be investigated.

COL. HILL: That's exactly the direction Dr. Pilmanis is moving. It's going to be very complicated and it's going to be time consuming to complete.

DR. PILMANIS: What we were talking about is an interactive physiological matrix. Call it model, but I think you need to start looking at individual physiological parameters, determine the varied interactions and establish risks. Clearly, loss of consciousness is your number one concern. Having a knee pain is really not a major issue for an individual in an emergency situation. Hypoxia and loss of consciousness is most assuredly your main concern at high altitude. Would you agree?

LT. COL. DEMITRY: Yes.

DR. PILMANIS: You now have an OBOGS system that's not giving you 100%, so breathing pressures may have to be higher than what would be needed if you were breathing 100% oxygen. The concentration may even be 70, 80, maybe 85%. It depends on what OBOGS delivers. Is it possible that the concentrations may drop below 90% at 60,000 feet?

MAJ. MILLER: Yes, it's possible. If the system is working correctly, you're going to probably get 93%. The possibility exists that the monitors may allow the concentration to drop to 90% or slightly lower before detecting the condition. So the concentration may be as low as 89%.

DR. PILMANIS: The pressure breathing schedule must then be established.

COL. HILL: You're not going to answer these questions today.

PROF. ERNSTING: Could I raise a question? I think you have to define what sort of decompressions you're talking about because there are lots of other things that happen in the environment. What happens to the cabin temperature? How big is the hole? Where is it? Are we just talking about the dump valve sticking open or are we talking about some failure of the structure due to enemy activity? Very often temperature might be the reason for descent. And how does the ECS work when the cabin altitude is 60,000 feet?

COL. HILL: You may have even encountered complications before attaining 60,000 feet. At least we're listing the factors.

LT. COL. DEMITRY: We can make this so complex that pretty soon there's no reason for me to be there. I'm already fighting to stay in my airplane anyway. There are plenty of guys out there, not so much in life support, but in propulsion, flight controls, weapons and avionics that want me out of my cockpit anyway. I'm trying to help you guys keep me in there so you need to give me some rules to follow after loss of pressure. It is like a Nintendo game that I play up there, and some people are saying I could be doing it with a datalink far away. We don't quite buy that yet, but you guys may get me dangerously close. So I need to help you give us the answers.

I still need some rules of thumb and I think we can make some assumptions. You will likely start from an initial set of conditions. As a result of indicator tolerances, the starting point may vary slightly, but if things aren't in my favor I'm not probably going to complete the mission. The airplane is valuable, no matter what airplane we're talking about, and I'm going to try to get the airplane home to fix it so that I can come back and do the job. I'm not interested in heroics. So we're going to have a starting point, and we may need to make some reasonable assumptions, but the user still needs some simple safety rules to follow.

This other thing is risk. As a flight surgeon, even as a pilot physician, I don't know if there is an acceptable risk of DCS. You see DCS in the laboratory a lot more than flight surgeons in the field. What is the acceptable risk of DCS? The answer to this will frame the approach to take in future DCS studies. Realizing that you can't make the decision in a vacuum, it would be appropriate to provide information on accepted levels of risk to the ACC and the

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program offices to assist policy makers in their decisions. Is that reasonable, or do you already have a well accepted level of risk that you're working with in different countries?

DR. PILMANIS: Let's look at what has been accepted. As I mentioned a few minutes ago, the U-2 has been flying with a level of 60-70% DCS risk for more than 30 years. It's been accepted operationally.

LT. COL. DEMITRY: That stems back to critical operational requirements. Your research is bringing what has been acceptable to the conscious level. I'm not sure that the risk was understood earlier.

DR. PILMANIS: You can assume that a higher risk is acceptable in an emergency situation than under normal operating conditions.

LT. COL. DEMITRY: I as an operator would agree.

COL. SHAFFSTALL: The primary option following loss of pressure at high altitude, at least in training flights, would be to descend quickly?

LT. COL. DEMITRY: There is nothing in peacetime that would warrant me to remain at high altitude following loss of pressure. We may train some emergency profiles, but my recommendation wouldn't be that you have to shut the engines down to really get the full training effect of it. We're going to come down and I know you agree with that.

COL. SHAFFSTALL: The protective system still needs to be fully functional because loss of pressure will likely happen more often during training.

LT. COL. DEMITRY: You're absolutely right. If we're just crossing a SAM belt at Mach 2, it's not going to take very long. You really would have to be unlucky to have a catastrophic failure of the pressurization system. This is going to happen much more in peacetime doing routine kind of operations and practicing, so we're in total agreement. Dr. Ackles, would you now make some leading comments?

DR. ACKLES: I think Col. Demitry reviewed the scenarios very well. Its appears that it is generally agreed that in peacetime the current technology get-me-down assemblies should provide adequate protection for emergency descent. However, we also need to prepare for a scenario where you will be exposed for an extended time at higher altitudes. I feel that each country represented here has much of the information that will be required to answer the question. One of the challenges is to put this research data together.

One of the first things we need to do is establish our research priorities. I agree that there is going to be a risk of decompression sickness following a loss of pressure at high altitudes. We will most likely be required to accept a certain degree of risk in this condition, but we don't currently have the data to establish the level of risk.

There are several issues that have come up at this meeting that need to be immediately resolved. For instance, the question of the importance of atelectasis versus 100% oxygen. That has implications that impact everything we've been discussing relevant to high-altitude exposure. With respect to OBOGS operations, I think we must expect that the pressure breathing schedule must be increased to compensate for the lower than 100% oxygen concentration produced by the system. We must determine the exposure altitude(s), whether you need to positive pressure breathe and then decide what the oxygen concentration/pressure breathing schedule should be, e.g., in Canada, we've accepted absolute lung pressures of 100 mm Hg for emergency loss of cabin pressure. I would not recommend that if I was going to stay there for a few minutes. Others may prefer higher levels of PPB, but we've got to look at all the factors and make compromises. The compromise may not be optimal, but might be acceptable under emergency conditions.

We have a lot of information on the effects of long term exposure to high levels of PPB, i.e., 10 and 20 minutes, which many would have thought was ridiculous a few years ago. With improved suits, we have even demonstrated

that subjects can pressure breathe for 20 minutes in 88 mm Hg. We had mixed results at this level of PPB at altitude, so we will be required to repeat the studies with different G-suit to vest ratios.

One of the things coming out of this meeting is that we jointly have a lot of information; the DCIEM at Toronto, the RAF at Farnborough, here at Armstrong Laboratory as well as other research organizations. If we're going to make significant progress resolving problems in this area, we have to come to an agreement to seriously share our data bases, rather than giving interesting papers at a meeting and then returning home to work on our internal research efforts. Much of the reluctance to agree on various topics is because the methodology, special details and data are not available for all to review. It would be really good and progressive to actively share our data bases. Otherwise it will probably be a long time before we can answer many of these questions. We've all got part of the answer. It's a matter of getting the working level people together and sharing a common data base before all the gaps in knowledge can be isolated and resolved.

I think we can agree that 5 minutes or less at 60,000 feet is attainable using current concept life-support equipment, pressure breathing schedules and trousers/vest ratios, i.e., as opposed to the traditional 30 seconds. This would likely require 75 mm Hg PPB with the 93% OBOGS product gas. We may even be able to improve the altitude exposure limit is we relook at the increased coverage G-suit and vest PPB ratios at altitude. The 4 to 1 ratio that was initially established for the standard G-suit is not as effective as lower ratios using the full-coverage suit. There are still several things about the data that needs to be sorted out to completely explain differences obtained in the study, e.g., why TLSS equipment fared better at the 4 to 1 ratio, ventilation/perfusion factors, etc. That completes my thoughts on what we need to do to accomplish our goals relevant to high-altitude exposures.

DR. PILMANIS: I think you would impose a very high risk of DCS during an exposure for 5 minutes at 60,000 feet, especially if you have earlier been exposed for a long period to a cabin pressure of 22,500 feet. Maybe that's okay?

LT. COL. DEMITRY: From the user perspective, it may be okay, if it's not a central nervous system (CNS) hit.

DR. PILMANIS: That's right.

LT. COL. DEMITRY: We really need the risk analysis to delineate the possibility as well as type of DCS. The type we really care about is CNS or chokes and whether it's going to compromise one's ability to recover the airplane. Other than that, you might be able to coax me into tolerating quite a bit of discomfort and pain from simple bends.

DR. PILMANIS: That's right, keeping in mind this is an emergency scenario. The risk of the more serious symptoms of DCS, however, is impossible to define. From previous data we can tell you that it is quite low, but for a specific altitude and time, it's impossible to establish the risk in any generic sense.

LT. COL. DEMITRY: Is it possible to come up with risk factors, like in coronary artery disease? Does anything correlate like the amount of nitrogen in the tissues?

DR. PILMANIS: The thing that works against the fighter pilot, unlike the U-2 pilot, is that there is inadequate time to preoxygenate. With little or no preoxygenation, you will have a higher percent of the more serious symptoms of DCS. From retrospective data, it appears that the U-2 pilots experience about 13% CNS symptoms. You're going to have a higher risk with zero preoxygenation, but I can't tell you what it might be.

COL. HILL: Further, the onset time at high altitude may be very, very short. What you can't answer is whether the probability of the seriousness is also increased.

DR. PILMANIS: I would assume at 60,000 feet, with no prebreathing, you might expect a DCS symptom within minutes.

COL. HILL: It should also be noted that the full-pressure suit now being used will maintain the U-2 pilot at a physiological altitude of 35,000 feet while the older partial-pressure suit would maintain him at 40,000 feet.

DR. PILMANIS: That's right.

COL. HILL: But now we have a fighter pilot potentially flying with a cabin pressure at or above 60,000 feet without a comparable pressure suit.

LT. COL. DEMITRY: You're making a very strong and persuasive argument for resolving the atelectasis and the 100% oxygen question.

COL. HILL: That's right, that's the basis for it.

LT. COL. DEMITRY: What are the clinical sequelae, the operational sequelae of atelectasis? I'm still unclear, having seen some very nice papers, as to atelectasis; what does it mean--not only acutely, but what are the long term effects? Is it possible to add a little positive end expiratory pressure (PEEP) to clear the airways? Am I doing something to the elastic membrane of the lung that will give me trouble after repeated exposures?

MAJ. DIESEL: It should be remembered that not only are we going to 60,000 feet, but we're going there predisposed. There may already be formed bubbles and aircrew do fly with bends all the time. If you're flying with bends and you suddenly lose cabin pressure at 60,000 feet, the situation will likely become forbidding.

DR. PILMANIS: That's what I tried to point out with the venous to arterial cross-over potential to indicate that we really don't know what the risk will be at 60,000 feet. We know what the risk will be at 30,000 but not at 60,000 in the current concept limited coverage partial-pressure suit.

COL. HILL: I'm afraid I know the answer, but we need to ask the F-22 SPO, and perhaps ACC/DR, whether a change in the pressurization schedule would be acceptable? I'm fairly certain that you can't structurally maintain a 7 PSI differential, but just changing the pressure a little might significantly decrease the risk of DCS while maintaining maximum altitude. Is there any possibility of maintaining a 5.5 or 5.75 PSI pressurization schedule?

MS. MCGARVEY: No.

COL. HILL: Okay, that's what I was afraid you'd say.

DR. PILMANIS: You have to stay with 5 PSI differential?

MS. MCGARVEY: Yes.

DR. WEBB: We are also required to breathe a variable (small) quantity of nitrogen in MSOC product gas.

DR. PILMANIS: It predisposes to bends.

MS. MCGARVEY: As a result of the reliability of the pressurization system, the chance of us having a rapid decompression at 60,000 feet is extremely low unless by enemy action. Beyond that, the pilot will be warned early enough that he can make a decision to descend to a lower altitude.

COL. HILL That isn't true for other aircraft. I worked on the canopy-off program for the F-15 and 200 canopies were lost over a 10 year period. I don't know what the failure rate is now, but it's probably not zero.

COL. SHERMAN: I don't remember any rapid decompressions in the SR-71 in my eight years with the program, but those are very small numbers of airplanes.

MS. MCGARVEY: If you look at the last 15 years of F-15, F-16, almost all the associated decompressions have been leaks that have occurred from ground. I mean leaks that were present in the pressurization system when he left the ground. We were only able to detect them after attaining 25,000 feet.

COL. HILL: In 1988, we lost the first pilot from DCS since the early 1950s. His case was mismanaged all the way, but his death was attributed to DCS. The cabin altitude was 28,000 feet.

DR. ACKLES: If the MSOC controller was set to maximum in the F-22, would it be capable of consistently producing 93% oxygen or are there other factors that affect it?

MS. MCGARVEY: The higher the ventilatory demand that the person places on the system, especially at the lower altitudes, the lower will be the capability of the MSOC to maintain 93% oxygen..

DR. ACKLES: The normal demands of a fighter pilot would be adequate to reduce the concentration?

MS. MCGARVEY: Yes, especially when pulling high +Gz. A 50 liter per minute flow at a very low altitude will reduce the concentration to as low as 60 or 70%.

DR. ACKLES: This is including the Positive Pressure for G (PBG)?

MS. MCGARVEY: Yes. Those are the conditions where we have the greatest demand on the system in terms of flow rate generation. Under a much lower flow rate, the concentration will probably be in the 90-93% range.

DR. ACKLES: So you can't maintain a 93% OBOGS product gas during all flight conditions?

MS. MCGARVEY: Not at low altitudes.

PROF. ERNSTING: You can retrospectively, if you increase the size of the OBOGS bed.

MS. MCGARVEY: That's true.

PROF. ERNSTING: Although I was responsible for the ASCC minute volumes which were used, we have recently found, by measuring minute volumes on the centrifuge under pressure breathing and G, that we're nowhere near the 50 liters per minute volumes. Pulmonary ventilation while pressure breathing at +9 Gs were running around 20 liters per minute. We were measuring flows both to the mask and to the garments during these studies.

DR. ACKLES: We find also that the greatest instantaneous flow demands are when you're doing the M1 maneuver, not during PBG. So maybe you will be better off at higher altitudes.

MS. MCGARVEY: There are other conditions that you can draw 40 or more liters through the system, but your mean concentration will likely remain much higher than the 60-70% level at the lower altitudes.

DR. PILMANIS: So is it fair to say that under most conditions of flight that we will have to accept 93% or less oxygen, that is unless we activate the emergency cylinder?

MS. MCGARVEY: At altitudes higher that 20,000, the OBOGS will most likely produce 93% oxygen. The chance of the system actually producing 93% on a steady basis is very, very good. However, we don't have a lot of service life data on OBOGS to firmly attest to its long term characteristics. It could be that the lower oxygen concentration range is absolutely insignificant because the OBOGS just doesn't descend into those ranges during its service life.

LT. COL. DEMITRY: That's a key operational issue to pass on to the users, i.e., to ensure that they have the information they need following a loss of cabin pressure. They may need to return to much lower altitudes; that kind of information is very important to make sure it gets into the checklist.

DR. PILMANIS: Keep in mind that if you drop below a concentration of 90% oxygen and argon (balance 10% nitrogen), you're really not doing much good as far as denitrogenation is concerned. That's supported by minimal research, but that's the way we view it.

COL. HILL: Regardless of the altitude?

DR. PILMANIS: The work was conducted only at ground level.

DR. WEBB: The scenario that Col. Demitry used involved a normal start, taxi, takeoff, and climb-out. You will not be pulling any Gs?

LT. COL. DEMITRY: No.

DR. WEBB: So you should be getting a reasonable MSOC gas mixture.

LT. COL. DEMITRY: Right.

DR. WEBB: If you were to experience a rapid decompression you're more than likely not going to engage?

LT. COL. DEMITRY: I'm not looking for a fight at that point. If my adversary were to let me alone, I'm happy to leave him alone.

DR. WEBB: Then you will descend to a lower level to avoid hypoxia and DCS?

DR. PILMANIS: I think we can agree on that.

LT. COL. DEMITRY: But beyond that, I'm trying to manage the risk between the physiological hazards and a successful enemy engagement. I am not trying to eliminate risk. I accept that there will be risk. I will have a good idea of what the enemy can do to me; you must give me the other half of the equation regarding what the hostile environment can do to me.

COL. HILL: Earlier, Ms McGarvey noted that the chances of decompression are low. I don't know whether a risk analysis has been conducted on loss of pressure in the F-22? Track records are available for both the F-16 and the F-15. I don't know if the F-22 has a significantly different pressurization system. If it's similar, then we probably have a kind of a track record. Have you ever experienced a decompression in the F-16?

LT. COL. DEMITRY: I flew some of the oldest ones and I would say that all the problems that we had were gradual leaks and I would agree with Ms McGarvey's assessment that I probably took off with it that way.

COL. HILL: I was just curious. Do you know anyone who has lost pressure in flight?

MAJ. NEUBECK: The only rapid decompression that I know of was a canopy loss.

COL. HILL: I understand. After we did the canopy-off tests in the F-15, the Systems Office indicated that it should never happen. Within a month they lost one at 1.7 MACH . But you're right, the decompression usually occurs very slowly, unless there's a catastrophe.

LT. COL. DEMITRY: That's true.

MAJ. NEUBECK: In an F-15, if the canopy seal wasn't working, you generally knew it on the ground. The canopy would rattle around and you checked it out before take-off.

COL. HILL: It would be nice if we could be provided the data.

MS. MCGARVEY: We gathered decompression data from Norton in 1989, identified and pursued fixing the failure modes for the F-15, F-16 and F-4.

LT. COL. DEMITRY: Realize that decompression rates are usually under reported and the prevalence and incidence is going to be significantly higher, but perhaps not as regards a physiological incident.

MS. MCGARVEY: The other failure breakdowns, that we know about, are on the environment control system, where the air cycle machine seizes up and we know how often that happens. When it does happen you lose pressure gradually and it is detected early on. We have some valves that potentially could have failed closed, but we now have them failing to an open condition. Those problems were seen in F-15s and F-16s. We separated the life-support systems from those failures.

COL. HILL: I think we can accept that the probability is not zero. Do you think it's much lower than previous aircraft?

MS. MCGARVEY: I think we will still see gradual ECS pressurization losses or breakdowns in the ECS system. They will probably be equivalent to what we see in the 15s and 16s, internal to the ECS. But I think we've done a better job of fixing the canopy seal problems. We will detect the gradual leaks early on.

LT. COL. DEMITRY: I really am not specifically addressing the F-22, but do you have a dump port in the cockpit?

MS. MCGARVEY: Yes.

LT. COL. DEMITRY: One still has to worry about failure modes of that port, or even a human error. I would imagine that it would be detected early on, unless it was accidental.

DR. PILMANIS: What are the expectations of decompression in the Eurofighter?

DR. MACMILLAN: There is a possibility of decompression while pulling G, certainly in the development aircraft, simply because the airframe is flexible. But the risks of decompression at very high altitude I don't think are any greater from non-enemy action than there would be in any of our current aircraft.

COL. HILL: We decreased the complication if we can assume it's near zero.

CAPT. O'CONNOR: What if you do lose pressure after passing over a SAM belt to the target area, the aircraft is still flyable and your only way back home is the way you came in, don't you still want a life-support system that will provide you the protection to let you go back high?

MS. MCGARVEY: Let's ignore the fact that it's a rapid or gradual decompression and he's still over enemy territory, can he still stay high?

DR. PILMANIS: Your real concern is loss of consciousness. Can we agree on that? That really takes us back to hypoxia and pressure breathing syncope. I'd personally accept the risk of DCS under those circumstances, especially if my only other choice was to get shot down and I knew that a few minutes would take me out of the area of greatest hazard.

CAPT. SCOGGINS: I would think you would want to be much beyond the level of mere consciousness in today's high-performance aircraft, especially if you're in a compromised condition and you've got to go through a high-threat area to get back home. I think we are now starting to look at an operationally relevant performance matrix, beyond the level of barely keeping somebody awake and breathing.

DR. PILMANIS: I'm afraid the only way you can give him better protection at 60,000 feet and above is to use a full-coverage partial-pressure suit.

DR. MOON: I think all of the data that I've seen have, in terms of rapid decompression and the degree of oxygenation, been in normal people with normal lungs. However, if one experiences a rapid decompression, having just completed some high-G maneuvers, those lungs are not going to be normal and, therefore, the allowable

ceiling where consciousness or reasonable psychomotor performance will be achieved is going to be much lower. It may be worthwhile to conduct an experiment to determine oxygenation after rapid decompression following G maneuvers.

LT. COL. DEMITRY: I don't think aerodynamically the airplane has the lift to generate more that 3 Gs at altitudes above 40,000 feet. That's not to say the next generation propulsion system may not take us there, but I can't imagine more than 3 Gs ever at 60,000 feet.

DR. PILMANIS: I had understood that atelectasis starts around 3-4 Gs and that someone had indicated that future aircraft would be able to pull up to 4 Gs?

MAJ. NEUBECK: You can get 3 or 4 Gs at 50,000 feet. That's about all you're ever going to get and you will lose energy fairly fast.

DR. SEARS: There seems to be a philosophy difference between the Eurofighter 2000 and the F-22 from the standpoint of emergency oxygen systems. The F-22 has an emergency oxygen system that contains only 100 liters, which effectively limits the amount of time you may remain at the higher altitudes. The Eurofighter has a 200 liter system.

MS. MCGARVEY: Depending upon the altitude, and whether the concentration is adequate we are planning on using the 93% MSOC system.

DR. SEARS: The MSOC will function adequately at 60,000?

MS. MCGARVEY: Yes.

MAJ. CAULKINS: There may be an operational issue during loss of cabin pressurization at 60,000 feet. If the OBOGS fail light comes on, I would activate my emergency oxygen. Once you've activated the emergency oxygen, there is really no way to turn it off. Even if the OBOGS fail light goes off, you will use up your emergency oxygen system over time.

MS. MCGARVEY: Let's assume for some reason that you do use the emergency oxygen system. The pilot would be trained to use a different set of safety procedures following activation of the emergency supply at higher altitudes. The problem is that we don't have any of the equipment to allow me to tell you the concentration will be 93% or that the regulator provides adequate pressure, etc. It will be a year or so before we can provide performance test data. It will then be another year before the aircraft system specific data are available. I think most will accept that 60,000 feet is a reasonable altitude for us to be with the current concept assembly and a 93% oxygen system. It's something we accepted and validated in both the YF-22 and YF-23 test program. Our initial plan was to have the pilot descend after 1 minute exposure at 60,000 feet to give him the opportunity to take whatever measures he needed to perform before descent. The questions remains on how long we expect to loiter at 60,000 feet, with 93% gas and 70 millimeters pressure?

DR. PILMANIS: I think we are pretty well agreed that one minute is not a problem at 60,000 feet. I don't know that we can agree on how many minutes you can safely remain. I'm not sure that we know.

DR. ACKLES: In breathing a 93% concentration at 70 mm Hg at 60,000 feet, you are walking very close to the edge. If you were to redesign your regulator for a longer period at 60,000 feet, I would have opted for a higher PPB level at altitude.

DR. GOODMAN: Of course, if the decompression were very gradual, you may be below a critical pressure breathing altitude by the time cabin pressure is totally lost.

LT. COL. DEMITRY: Yes, and then you could leave the area.

DR. PILMANIS: It's a reasonable scenario.

DR. GOODMAN: It certainly changes the philosophy regarding hypoxia, DCS, atelectasis, etc.

LT. COL. DEMITRY: That's a very compelling scenario. The problem is that there's so many variables, e.g., how fast is gradual, how high can he safely remain, how long? There may be several rules of thumb necessary?

DR. GOODMAN: An automatic controller that changes oxygen concentration, pressure breathing schedules, pressure differentials, and other critical variables as necessary for optimum protection would be nice.

LT. COL. DEMITRY: But until that day, we still need the exposure limits for each variable.

COL. SHAFFSTALL: Even then, you are probably going to push the limits until the symptomatology changes your mind.

LT. COL. DEMITRY: I don't know how to measure gradual degradation in cerebral function. I'm not sure that you will be able to hang in there until the symptoms get so bad as to warrant a descent. If there were a reliable means of distinguishing an impending loss of consciousness, then I would totally agree. However, since there are several conditions where loss of consciousness occurs faster than self awareness, pushing the limits may be self defeating.

DR. PILMANIS: I agree. I think you have to expect a much more sudden loss of consciousness at these higher altitudes.

LT. COL. DEMITRY: In case the problem is DCS, do you always experience some symptomatology on your way to loss of consciousness? I have never heard that was a requirement.

DR. PILMANIS: No.

LT. COL. DEMITRY: Of course not.

MAJ. NEUBECK: As regards DCS, when does loss of consciousness most likely occur? Could it be immediate or at some later time?

DR. SEARS: I can provide some information in this area. I was the subject on nearly all of Dr. Balke's bends runs in the early 1950s. We used to bend so badly after exercising without prebreathe at 38,000 feet that we couldn't get the chamber door closed to descend, i.e., depending upon the level of pain at the bends site, in many cases it was nearly impossible to use one or the other of your arms. In all these studies, I remember only one case of chokes, but we did have many cases of severe bends. I can't remember any unconscious episodes during the research studies, but do remember several CNS related unconscious episodes that happened during standard physiological training to 43,000 feet without any preoxygenation in the early 1950s. Unconsciousness occurs quite quickly when it does happen, but it doesn't occur very often with DCS.

PROF. ERNSTING: Our experience with pressure breathing syncope at altitude is that it occurs fairly rapidly. In my personal experience, you may get some of the usual symptoms of fainting, but it's usually fairly quick. I wouldn't like to push the system. In the late 1950s we were quite successful for at least 6 minutes with pressure breathing systems at 56-60,000 feet. So I don't have any worries about the 5 minutes at 60,000 feet and I suspect Alistair doesn't either, especially with current uniform counter pressure garments and particularly with 2 times G-suit pressures.

DR. PILMANIS: Dr. Sears, would you relate some of the early training profiles for the U-2 pilots?

DR. SEARS: Yes. U-2 pilots were trained early on in the MC-3 and MC-4 capstan partial-pressure suits. The profile included a 2 hour denitrogenation period at ground level followed by a 2 hour exposure to 65,000 feet. It

should be remembered that the suit fully covered the pilot to include pressurized gloves and helmet. We had few problems during these training runs.

In B-58 capsule studies in the 1960s, we exposed ourselves and crewmembers to a 3 second decompression to 60,000 feet with immediate descent to 37,000 feet in the enclosed capsule, wearing only an early model pressure demand mask that would contain only 30 mm Hg, i.e., no pressure vest or trousers. I don't have any major concerns either, for 5 minute exposures to 60,000 feet in the current concept assembly.

DR. GOODMAN: I understand that there are new technologies in chest mounted Dopplers which could provide hands off Doppler monitoring.

DR. PILMANIS: I would strongly suggest we don't go in that direction. That has been tried and you cannot correlate bubbles with symptoms. We have now covered the sustained operations question. I would like to thank the panel moderators and participants for their input on the topic of sustained operations.

Workshop Discussion, Conclusions and Recommendations

MODERATOR: DR. ANDREW PILMANIS, AL/CFTS, BROOKS AFB, TEXAS

DR. PILMANIS: We've had a couple of interesting days. This session is devoted to bringing it all together. This is likely the most important part of the workshop. One of the things that was addressed several times yesterday was the question of acceptable risk. Four years ago, some of you attended a workshop here on altitude DCS. If you don't have a copy of the proceedings, I'll be happy to give you one. One of the statements in the executive summary from that workshop was, " it was pointed out that the definition of acceptable risk varies with the mission and ultimately must be decided by the operational people responsible for that mission. It is the responsibility of the investigators to equip these field managers with the best available research information to enable them to make informed, rational decisions. In turn, the investigators must have access to accurate and complete feedback from the field in order to frame the research in proper perspective." I think that still applies.

LT. COL. DEMITRY: Those proceedings were how long ago?

DR. PILMANIS: Four years.

DR. PILMANIS: What we will attempt to do this session is to review each of the topics covered in the workshop and come to some sort of an agreement and/or recommendation for further discussion and study.

To start with, it is my understanding that the F-22 design has been finalized and, therefore, there is no immediate need to resolve the acceleration atelectasis problem. The MSOC switches to high cycle above 11,000 feet and there is no way currently to accept any change toward a higher oxygen concentration to reduce the possibility of DCS. Apparently, the Eurofighter 2000 design is also fixed, and even if we had another answer today it probably wouldn't change things. So it's really a longer term question. Can we agree that a definitive study is needed that will establish the effects of assisted positive pressure breathing on acceleration atelectasis? We don't have that data.

PROF. ERNSTING: I would certainly support that, yes.

DR. PILMANIS: Any other comments on that topic?

LT. COL. DEMITRY: I would also be curious, from an occupational health standpoint, what the long term effects of repeatedly collapsing a lung might be? Are there any chronic problems 10-15 years out for these aviators? Does it do anything to the elasticity of the tissue? I can't envision that that would happen, but a long term strategic view of the health of the aviator should be part of the study.

DR. PILMANIS: Should we ask the Navy to look at their fliers for the last 20 years?

LT. COL. DEMITRY: It would be very difficult to come to any definitive conclusions from a retrospective study. I suggest that we put the surveillance tools in place and go forth.

PROF. ERNSTING: I was going to support going to the United States Navy, but some of their aviator pulmonary studies were conducted before they started using 100% oxygen. As you know, they did very intensive long term studies in the United States Navy.

LT. COL. DEMITRY: And I don't think from our standpoint that the US Air Force will have the same kind of a problem based on where we're heading with pressure breathing for G.

PROF. ERNSTING: The other thing I didn't mention is that the RAF operated on 100% oxygen for three years in the late 1950s. We looked at the long term health risks and found nothing.

DR. PILMANIS: One of the things that was driving part of this argument is the DCS aspect of breathing 100% oxygen. It should be reiterated that when we talk about 100% oxygen, or MSOC product gas at full capacity (93%) as relates to prebreathe, they are equivalent as far as DCS is concerned. I want to make it clear that argon is not a problem. When you start dropping below a certain MSOC oxygen concentration and start increasing nitrogen in the mixture, that's when you're potentially going to run into denitrogenation problems; not with the 5% or so argon.

DR. WEBB: I understand there is a toggle in the F-22 to allow switching from either 100% product gas or normal oxygen?

MS. MCGARVEY: That's correct.

DR. WEBB: I would think it would be better to go to 100% product gas starting with the first checklist, assuming that they aren't expecting immediate aerial combat.

CAPT. O'CONNOR: When you noted the non-effectiveness of 90% or lower +oxygen for denitrogenation, were you speaking in terms of increasing the concentration of nitrogen to 10% or higher?

DR. PILMANIS: Yes. If you increase nitrogen in the mixture to 10% or higher, denitrogenation is basically ineffective.

CAPT. O'CONNOR: If your OBOGS nitrogen level is around 4 to 5%, it is within the acceptable range?

MAJ. MILLER: If you're running let's say 90% oxygen out of an OBOGS, you might get around 4.5 % argon.

DR. PILMANIS: So the balance is nitrogen?

MAJ. MILLER: Yes. The balance would be nitrogen.

COL. SHAFFSTALL: Do we have an adequate understanding of the time required for nitrogen equilibration with the lower partial pressure of nitrogen at lower cabin altitudes? As an example, if the cabin altitude is 20,000 feet for an hour or more, and a decompression then occurs, have you off-gassed enough nitrogen for any protection from DCS? Or do we know what point in time that might occur?

DR. PILMANIS: No, we do not know what that point is. As a generality, you are balancing two things, bubble formation and denitrogenation. Once the bubble forms it remains without resolution for long periods. The longer you remain at these intermediate altitudes without experiencing DCS, the fewer bubbles you're going to form. But you're always balancing those two. No, we do not have enough information to accurately state how many hours it would take to denitrogenate at each intermediate altitude before decompression to 50 or 60,000 feet.

PROF. ERNSTING: Can you provide a better quantification of nitrogen concentrations between 0 and 10% relevant to their value for denitrogenation?

DR. PILMANIS: Not really. We're on shaky ground in that area because there's only one paper that directly addresses the problem. The conclusion was that you effectively had no denitrogenation below 90% oxygen with balance nitrogen. The study needs to be conducted at various concentrations, e.g., it may be closer to 85%. It does make sense when you look at non-linear nitrogen dynamics, i.e., a 50% oxygen/nitrogen does not provide a 50% elimination of nitrogen. That's just not the case.

Let us now switch to the topic of positive pressure breathing. Some years ago there was a report out of the University of Southern California authored by Drs. Meehan and Henry. Dr. Henry, as Dr. Sears indicated earlier, developed the first capstan partial-pressure suit. Also, he conducted studies on pulmonary overpressure duringWorld War II. The USC study was designed to exercise the abdominal muscles for application to the weightless environment. Five subjects were trained in a stepwise manner to pressure breathe for 30 minutes at 60

mm Hg, four times a day, for 30 consecutive days without chest counterpressure. As one of the subjects, I can tell you that it was very uncomfortable but it can be done. And as has been pointed out repeatedly, training in pressure breathing is absolutely crucial. There were no associated medical problems in this study. I think it bears on the issue of whether a pilot will be able to pressure breathe at high pressures for any length of time.

DR. HARDING: Could you give us a demonstration?

DR. PILMANIS: Remember, it was over thirty years ago.

SQN LDR GRADWELL: But isn't there a problem here? You indicated that you were trained in a stepwise fashion over time. How many days of the week are the pilots going to be available to undergo pressure breathing training?

DR. PILMANIS: Training is extremely important. Pressure breathing training should become part of the training syllabus.

DR. STOLP: I think that's a very important issue. Training is important, but what patterns of breathing do you train? We now have evidence that with high pressures, we're getting close to the edge of what we can expect with pressure breathing so far as gas exchange is concerned. We know that it's not just the alveolar PO2, it's the delivery of oxygen to the tissues that's important. We may not want to just keep cranking up the pressure and spend more of our time trying to figure out how to minimize the cardiovascular effect. I would suggest that we also relook at other means to maximize oxygen delivery to critical organs, e.g., slowing the breathing pattern during pressure breathing may not be the correct thing to do when you start looking at arterial gases. We've gotten away with it earlier, but it may be more critical at these higher pressures.

DR. PILMANIS: I think it's important to note that each presenter discussing pressure breathing studies had some subjects that experienced syncope. That does indicate that we are close to the edge, maybe too close for fine tuning?

DR. STOLP: Maybe we ought to stop fine tuning and look at different methods of delivery. We need to maximize O2 delivery to critical organs and determining the optimal breathing patterns. Professor Ernsting noted that his subjects were not hyperventilating. To define hyperventilation we need arterial gases, we need to know where that gas is going and how it's being delivered to those critical organs.

PROF. ERNSTING: There is a vast amount of experience of pressure breathing at 30 mm Hg. We've conducted extensive cerebral function studies and know that if we push beyond 30 to 35 or so mm Hg at 50,000 feet, we'll get faints.

DR. STOLP: But we haven't conducted cerebral function studies at the higher pressures we're now talking about.

PROF. ERNSTING: No, but I've conducted arterial sampling during pressure breathing studies at 56,000 feet and our PCO2s were 30 mm Hg in subjects over a 5 to 6 minute period. I don't know whether anybody else has done that. We did that extensively in the 1950s before we put forward the jerkin, G suit, mask system. So there is a great deal of background experience on the effects of hyperventilation, either from measuring pulmonary ventilation or from actual blood gases.

MS. MCGARVEY: Relative to training, for the policy makers to make a decision on what their training syllabus is going to look like, someone needs to translate the positive pressure breathing for G (PBG) information to the altitude experience (PBA), i.e., differences or similarities in breathing patterns, rates, etc.

DR. GOODMAN: I would add that the training curve is really rapid. You can take a naive person and train them to properly pressure breathe within a matter of a few sessions. From my own personal experience, it's not a prolonged process. Once you get people on the learning curve, they can come up very quickly and keep the skills for a long time.

MAJ. CAULKINS: It's important for us to take into consideration that even if we were to determine that the pilot could positive pressure breathe at 60,000 feet for 10 minutes, the oxygen system, at least in the F-22, was not designed to allow him to remain at this level for 10 minutes. It was designed as a get-me-down system and, therefore, some fundamental changes would have to be made to the system to allow someone to remain there and positive pressure breathe for that period at 60,000 feet. If we were to use OBOGS product gas, we need to increase the breathing pressure schedule or we need to provide a larger 100% oxygen supply, either through the emergency oxygen system or a separate backup system. The system was just not designed to allow the aircrew to remain at these higher altitudes.

DR. PILMANIS: I think that's a very good point. You may need 75 to 80 millimeters, not 70 if you're going to stay longer.

MS. MCGARVEY: That's right. The intent of the F-22 life-support system is to provide a get-me-down capability and the Air Combat Command (ACC) did not express any interest in staying up high.

DR. PILMANIS: I want to again point out that this meeting was not specifically aimed at either the F-22 or the Eurofighter, but to set some goals for what may come downstream. Some of these discussions do apply directly to these aircraft, but we need to keep looking forward to provide research input for future needs or systems.

MAJ. NEUBECK: We're currently expecting to buy and produce 442 F-22 airplanes, but there will be derivatives of this aircraft that have the same capabilities, but different missions. If higher pressures or a greater oxygen supply is something that we ought to look at for future missions, then that's something that we need to take into account and put on the record.

DR. PILMANIS: That's a very important point. There are several changes that have been discussed in this workshop, e.g., providing an adequate supply for pressure breathing with 100% oxygen or raising the pressure breathing schedule using lower concentrations. Another change might include increasing the cabin pressure differential.

DR. ACKLES: I agree. If you're going to remain at 60,000 feet for slightly longer periods, and you're using OBOGS breathing gas, you had better count on pressure breathing at 75 to 80 millimeters.

SQN LDR GRADWELL: I think we would probably all agree that you don't want to pressure breathe to any higher pressure than necessary. It seems more appropriate to spare the individual having to breathe an extra 8, 9, 10 mm Hg by providing an adequate backup supply of 100 % oxygen that can be selected at high altitudes with the capability of returning to the MSOC product gas at lower altitudes. Additionally, this involves providing an adequate emergency oxygen supply over and above that required for pressure breathing at high altitude. If that's worked into the design, whether you're staying up longer or descending, you're actually reducing the level of pressure breathing.

DR. PILMANIS: The down side is that you are required to carry a larger oxygen supply.

DR. SEARS: I feel compelled to say this again. If you want to take care of most of the problems we have been discussing, have the aircrew wear a pressurized helmet for PBA with integral high-pressure mask for PBG, a sleeved vest and full-coverage anti-G trousers with a regulator that will provide the correct breathing and suit pressure schedule for either the altitude or the +Gz exposure. Then you will be able to provide 140 mm Hg pressures or more and remain at 65,000 to 70,000 feet for quite long periods without many concerns.

DR. PILMANIS: Since Dr. Sears has brought it up, I think it's worthwhile to consider all options. Over the past two days, we have discussed some elaborate research projects on positive pressure breathing. As long as the encumbrance of a full-coverage partial-pressure helmet remains unacceptable to pilots, there is considerable job security in the high-altitude research community. All would likely agree that you can more safely and comfortably remain at higher altitudes for longer periods in a partial-pressure assembly with an enclosed helmet. You could

even go safely to much higher altitudes and stay for longer periods. Currently, the pilot is limited by his protective ensemble. Provide the pilot with an enclosed helmet and the aircraft now becomes the limiting factor.

PROF. ERNSTING: I don't think it would do any harm to repeat that in modern combat aircraft of this generation and the next, the thing that's going to drive the need for positive pressure breathing is that required for increasing G endurance. We're going to be positive pressure breathing with G as long as we've got very agile airplanes and I can't see pilots accepting a pressure helmet. I think it's our task in the high-altitude emergency condition to extend the breathing systems as far as possible and accept the additional risk. It's very important for the altitude and acceleration groups in the laboratory to review each others needs, because the situation of pressure breathing under G is entirely different than pressure breathing at altitude, e.g., considering positive pressure breathing for G, the counterpressure bladders over the chest can be reduced because you've got 9 G forcing the chest downward, assisting in lung mechanics and that's probably not the right approach for pressure breathing at high altitude. In the RAF, our very clear mandate from the Air Staff is never to go back to using partial-pressure helmets in next generation airplanes.

DR. ACKLES: I agree with Professor Ernsting. We are really focusing on an optimal ensemble for PBG and then looking at the what we can do to provide pressure protection at higher altitudes, e.g., changing G suit to breathing pressure ratios, increasing pressure schedules, etc. That's a very important point.

DR. SEARS: I don't want to overstate the case for a pressure helmet, but when the COMBAT ACE system is produced, you will be wearing a fully enclosed helmet for chemical protection . The system will incorporate a high-pressure mask for PBG, a pressurized vest and full-coverage pressurized trousers. It doesn't take much imagination to move from that starting point to a helmet that would only pressurize upon decompression to higher altitudes. Indeed, the Northrop suit I mentioned earlier combined both acceleration and altitude protection. I would suggest that an integrated concept such as this could be improved on to the point that it would be acceptable to the aircrew. It will take time to iteratively improve the system, but I firmly believe that it can be accomplished. I also believe that in just a few years, we will need physiological protection to at least 70,000 feet for longer periods of time.

MAJ. NEUBECK: We started losing aircraft and pilots from high G in the F-16. We now have the F-22 that is really fast even without afterburner. So you have a lot of potential energy from the engine that can be sustained for a very long time. The current concept mask/vest/G-suit assemble will help the pilot survive and maintain those high Gs. I think we're going to be faced with the acceleration problem for a long time.

DR. PILMANIS: You won't let us get away with an easy solution, will you? Are there any other comments on pressure breathing?

DR. MOON: I think the simplest thing about positive pressure breathing is that it does work. Several presentations have shown that you can support oxygenation at high altitude. But there are some problems. The degree to which PPB may normalize brain oxygenation in particular is limited by cardiovascular effects, hyperventilation and to some extent by discomfort and clearly those issues have to be addressed. Furthermore, the achievable normalization of oxygen, at the highest altitude where you can keep a pilot conscious, may be attenuated by the small amount of nitrogen or argon in the breathing mix from any practical OBOGS system. I think it's also worth mentioning that the more successful anti-G garments might be expected to predispose to pulmonary barotrauma. Finally, the ventilation perfusion abnormalities induced by PPB indicate that end-tidal gas partial pressures are not good indicators of what's going on in the arterial blood. So use of end-tidal gases as end points in terms of oxygenation or PCO_2 during PPB are not likely to be very useful.

DR. PILMANIS: Other comments on positive pressure breathing?

MAJ. NEUBECK: Dr. Webb brought a chart in this morning that I hadn't seen before. Our impression was that if you were going to fly at 60,000 feet, you could stay there as long as you want to with an intact cabin pressure of around 22,5000 feet. Based upon Dr. Webb's data, I start running a risk of DCS based on the length of time that I'm at a cabin altitude of 22,500 feet. I hadn't understood that would be a real problem at these lower levels or that a longer term exposure at these lower altitudes would impact the exposure limits at 60,000 feet following loss of

pressure. We're now getting into risks and training issues that pilots are going to need to understand about the environment at 60,000 feet.

DR. PILMANIS: I think it's good to separate normal operations from the rapid decompression to very high altitude. I equate the rapid decompression with an emergency situation and the rules change. Is that a fair statement?

COL. HILL: Maybe we should look at it on a probability basis. The probability of a 60,000 feet exposure is very, very low. We may have many exposures a month to 22,000 and 23,000 feet. Even with a 10% probability of DCS, is the DCS likely to be severe?

DR. WEBB: We currently don't have the data to predict the severity. During our studies, we brought the subject down quickly after initial symptoms. We didn't allow the DCS to develop, so we don't know if and how fast the DCS progresses after the initial presentation.

DR. PILMANIS: Let me interject that this is new data. Dr. Webb only reported on this a month ago and the study is still ongoing. I emphasize that because we saw significant DCS in 10 subjects, we have increased the scope of the study and renewed the protocol for more subject exposures. This is a study funded by NASA. You don't normally see such a radical threshold in physiological studies, but that's indeed what the data showed.

DR. WEBB: We now have completed 12 subjects at 21,200 feet and 14 at 22,500 feet and the effect is still there. We are planning to expose an additional 20 male and 20 female subjects.

DR. PILMANIS: And the effect is still there. How many total subjects will be studied?

DR. WEBB: 20 males and 20 females.

DR. PILMANIS: If the effect continues to hold, it's a very important finding. We're a little hesitant to jump on this with both feet because of the numbers. Keep in mind that the time factor is extremely important. If you stay only an hour, the risk of DCS will be low. If you remain at these cabin pressures for 2 or 3 hours, you will likely be exposed to the full effect. If you keep the time short you can get away with all kinds of things, e.g., training chamber flights to 43,000 feet have been experienced by thousands and thousands of aircrew with only rare DCS symptomatology.

DR. GOODMAN: Another point to consider is the decompression rate. If it's rapid versus slow, then it may changes these issues.

DR. PILMANIS: Yes, but I was only discussing routine pressurized flight without loss of pressure. As Col. Hill indicated, this will happen much more often in day to day operations.

COL. SHAFFSTALL: The long exposure time at these altitudes should be emphasized as strongly as the incident rate. It should be remembered that the Air Force has long term experience flying to 25,000 feet in unpressurized T-37s. The operational community will be hesitant to believe your results unless they fully appreciate the exposure times used in the study.

DR. PILMANIS: Yes. Exposure time at altitude is extremely important.

DR. ACKLES: What difference does it make, with just 30 minutes preoxygenation with either 100% or 93% MSOC product gas. Would that make a significant difference relative to DCS?

DR. PILMANIS: We made the assumption fighter pilots do not normally prebreathe.

DR. ACKLES: Yes, but if the pilot wanted to fly a high-altitude insertion mission, started breathing maximum OBOGS product gas on take off and it takes him 30 minutes to get to the insertion point, would that make any difference with DCS risk?

DR. PILMANIS: It probably would make some difference, but we don't have that specific information in our database. We would expect the curve to shift a little but not a great deal, unless you completed 1.5 or 2 hours of prebreathing. A half hour, or even an hour at these lower cabin altitudes, would probably not shift the curve very much. It has a much greater positive effect if you are subsequently exposed to higher altitudes.

LT. COL. DEMITRY: I take some exception with the comment that fighter pilots won't prebreathe. If you can show me that it's to my advantage to prebreathe, I will be interested. I am always interested in something that will allow me to out perform the other guy. I know guys who prebreathe for night missions because they read somewhere that 100% oxygen will increase their visual acuity. And these guys swear by it, especially the smokers. If you can show them that their performance will be degraded without prebreathing, they would most likely prebreathe.

DR. PILMANIS: I was thinking of the wartime scenario where you may be forced to get off the ground in a hurry.

MAJ. NEUBECK: The F-22 more than likely will go to a tanker for fuel which nominally takes 45 minutes. The flight to the operating area and the climb/cruise profile to get to high altitude will take another 1.5 to 2 hours.

DR. PILMANIS: That's the reason Dr. Webb presented the inflight denitrogenation data. If you can keep the cabin pressure at or below 16,000 feet, and are breathing a gas low in nitrogen, you will be effectively denitrogenating.

LT. COL. DEMITRY: Again, before the operators can make a decision, we need some sort of a model for selecting an optimum profile. You're going to have to construct these kind of charts for a matrix or a model with some sort of validity to allow extrapolation between data points to be able to facilitate smart tactical decisions.

DR. PILMANIS: We are currently pursuing a DCS model that, if it's successful, could go in the cockpit and give you immediate read-out, but that's some time downstream.

LT. COL. DEMITRY: So are these systems.

PROF. ERNSTING: Were the subjects breathing 100% oxygen from ground level?

DR. PILMANIS: Yes.

PROF. ERNSTING: How would it change the findings if they were breathing a 50% nitrogen mixture?

DR. PILMANIS: Quite a lot. It will significantly shift the curve towards a higher risk of DCS. Using our current diluter demand systems, inflight prebreathing will be ineffective. If you're in a situation where DCS is of concern, you should not be on a standard diluter demand system.

COL. HILL: OBOGS provides more than enough capacity to use 100% product gas all the time, correct? Of course it does. Except for the atelectasis and delayed ear block problems, you don't really have to dilute the product gas.

MS. MCGARVEY: It still depends upon the ventilatory demand on the system, which may lower the concentration at the lower altitudes.

COL. HILL: Is acceleration atelectasis the primary reason for not using 100% product gas?

MS. MCGARVEY: Absolutely.

PROF. ERNSTING: However, with the current design of the concentrator in the airplane, coupled with high demands, you will receive quite a bit of nitrogen at lower altitudes.

MAJ. MILLER: In other words, you can provide 100% product gas undiluted, but there's no guarantee it's always going to be at 93%. It could be lower. Is that what you were asking?

COL. HILL: Yes, that's exactly what I was asking.

LT. COL. DEMITRY: How low?

COL. HILL: If your flows are very high you will receive some nitrogen in the mixture, even though you're receiving 100% product gas?

MS. MCGARVEY: Yes.

MAJ. MILLER: Yes, that's correct. We don't have the system specific F-22 MSOC data yet to answer the question regarding the concentrations under various demands.

COL. HILL: If you're conducting normal flight operations, just throwing switches and flying the airplane, are there any oxygen concentration problems?

MS. MCGARVEY: We will be able to answer the question when we receive the actual OBOGS for test. We will likely find that, at even the maximum concentration or maximum flow rate that can be expected, concentrations will be above 90%.

COL. HILL: That seems to be the critical number.

MS. MCGARVEY: If it's really necessary to have that percentage, you could rapidly climb to 9,000 feet where we fully expect that the concentration will be 90% or above. They can climb to these levels in less than a minute.

COL. HILL: But you're impacting operations.

MS. MCGARVEY: That's right. However, we do the same thing for negative G. The pilot designs his maneuvers around limiting negative G exposure. We give them enough information and they'll determine how they're going to employ the plane.

COL. HILL: I think the pilot might debate that. Everything needs to be automatic so he can respond to the battle.

LT. COL. DEMITRY: I agree, but in all fairness, we should have had today's wisdom back when these tough decisions were being made. So, it seems to me you're both right. I agree that that's not an optimal solution and I also agree that there are many necessary work-arounds that make life more difficult, but it can be done.

COL. HILL: But you think there's a good possibility that the MSOC may be better than we fear and that the bed capacity will be adequate?

MS. MCGARVEY: Yes.

MAJ. MILLER: Oh yes, it's possible. We just don't have the data right now. Keep in mind this concentrator is designed to be very, very small. It is a small unit, because of the compartment size.

COL. HILL: Is it the same problem in the Eurofighter?

PROF. ERNSTING: The concentrator is very similar in fact.

DR. PILMANIS: Once again, 90% is not the number to keep in mind. If you add both oxygen and argon you're effectively at 94% considering denitrogenation. I think 10% or greater nitrogen is the important number to remember.

COL. HILL: We need to capture these variables before we proceed too much farther.

LT. COL. DEMITRY: Before we leave DCS, in the past most air forces have grounded a pilot that experienced DCS in flight, especially a CNS hit. Is the risk data available that would support changing medical standards that would allow the flight surgeon to put the guy back in the cockpit, or does he permanently need to be grounded?

DR. PILMANIS: The number one conclusion of the hyperbaric workshop four years ago was that DCS should be decriminalized. In the past, in the USAF, a pilot would be permanently grounded if he experienced a DCS hit in flight. It goes without saying that few pilots ever reported an incident of DCS. That's why we completed three anonymous surveys on the U-2 pilots. The high DCS incident rate was no surprise. Everyone in the community knew it was happening, but it was the first time it had been documented. Pilots will generally not report DCS for fear of being grounded. That's perhaps an over simplification, but that's the case. The AF Instruction was changed last November to take away the penalty. It's one thing to change the paperwork, but quite another to get the people in the field to follow the recommendation and that hasn't happened. Changes to administrative procedures are now in the hands of the medical community and not at the research level.

LT. COL. DEMITRY: Does the data , in your opinion, indicate that once treated for DCS, the risk of getting DCS on subsequent exposures is the same as baseline, or can that be established?

DR. PILMANIS: There are at least two things to consider. One, I don't think anyone will disagree that there is tremendous individual variability in DCS. I have seen subjects predictably bend at very low altitudes and others that you have difficulty in bending at 40,000 feet. There's that much variability. The susceptibility is fairly consistent within any one individual. Secondly, the idea historically evolved that if you get a neurological hit once, you're more susceptible the next time. There is absolutely no evidence for that and I personally don't believe in an increase in susceptibility on subsequent exposure to altitude. Whether you experience neurological symptoms, chokes or pain only, and you're properly treated with no residuals, there is no change in the baseline. The instruction basically indicates that they should return to duty in 72 hours. They require a consult with hyperbaric medicine and, if it was a neurological problem, they must have a neurological consult.

COL. SHERMAN: We can get a copy of the instruction.

DR. PILMANIS: Dr. Ryles has recently written a paper reviewing the DCS symptoms in our database. We see DCS cases every day and the paper is titled, "Initial Presentation". In our studies, as soon as they bend we bring them down, so all we see is the initial presentation. In that complete database there is only one CNS hit, is that right?

SQN LDR RYLES: That's correct, yes.

DR. PILMANIS: And that includes several hundred subject exposures. In the research setting, you primarily see only pain and paresthesias. We really don't see DCS episodes involving unconsciousness. On the other hand, if you discuss DCS with hyperbaric medicine who receive all calls involving operational DCS, they see a lot of neurological cases. The major reason is that operationally the crewmember does not come down immediately. The disease has time to progress. By the time the Hyperbaric Medicine team see the problem, you have a very different picture. Time again is the element that has to be taken into consideration.

LT. COL. DEMITRY: You're still making it difficult for the advocates in the field to say that the research is convincing enough to take a less conservative approach, because operationally the experience is more severe than what you're seeing in your chamber. I realize I can't ask a researcher to sign their name on the bottom of a statement indicating this is dogma. But at the same time you have to characterize the risk and then let the user step up and accept his responsibility for that which is still uncertain. If we make the answer too amorphous, the user will tell you to return when you have more information.

DR. PILMANIS: That's exactly what we're trying to do. Dr. Webb passed around a table the other day of predicted risk. Whether or not you believe it, you can look up your DCS risks. We answer calls from the field every day and answer them using the numbers from our limited data base and from our personal experience. We placed the numbers that we do have in a reference table because the calls we receive are quite frequent.

COL. HILL: Lt. Col. Demitry, I think when you asked for a table that would provide for a series of steps to lower altitude, I probably didn't make my point very well. If you have just one interval you can provide some confidence to it with your confidence dropping at each subsequent level.

LT. COL. DEMITRY: I understand that and the only way to start adding that kind of confidence is to start a matrix of experiments that address the specific question. I'm not involved with the day to day research, but you normally are looking for that one set of conditions and then you come down. Operationally I think what we're suggesting is there may be the need for a cascade, so that's why perhaps some funding ought to be focused on that kind of a product.

COL. HILL: I will do everything I can to make sure that happens. Its not appropriate for Dr. Pilmanis to comment on past support for the high-altitude research program, but we have experienced problems in the laboratory in that area. In addition to collecting information from other nations, Dr. Pilmanis has revolutionized the research approach, but it's taken a long time and it's going to take a lot more time to collect the data we need. That's unfortunate because we need many of the answers currently. If your frustration is that we don't have the data yet, it's well placed. Research is expensive and now it's difficult to find qualified subjects. I don't have the answers on how to get there in the immediate future; its too bad.

LT. COL. DEMITRY: Your point is well taken.

COL. HILL: I can't speak for Dr. Godfrey, but I can certainly speak for Mr. Brinkley and we intend to maintain this research at a healthy level because we think it's paying off. We'd like the using community, when you rack and stack things in the future, to put a kind word in regarding the need for information in this area every now and then, because there are a lot of other people out there with very sharp knives.

MAJ. KREBS: The best approach would seem to be to find an acceptable method to collect inflight data from aircrews. We lost a lot of applicable information in U-2 flights by the pilots not reporting DCS problems. We really lost the opportunity to change equipment and procedures that might have reduced their risk of DCS. If you look back to World War II, we had people on a daily basis experiencing decompression sickness. We had thousands of aircrew members and thousands of sorties and much of that data is gone. We didn't have the facilities to collect it as we do today. There should be some way to collect these data anonymously by downloading the postflight information to a research database with assurances to the pilot that it will not be seen by the squadron flight surgeon.

DR. PILMANIS: It becomes difficult when the problem may be career threatening to the pilot. He may not believe in the ability of anyone to keep the information anonymous.

MAJ. KREBS: If there were some mechanism for cataloging the number of sorties, altitude, time, duration, age, weight, and whatever parameters you feel necessary, and downloading the information directly to the laboratory without any possibility of tracing it, they might do it.

MAJ. NEUBECK: If you educate the flight surgeons and inform them how you guys feel about not grounding pilots, you'd move a step closer to what you're looking for. You're not going to convince any pilot to write anything down, especially since he's probably going to come in with a hood over his head and his name tag off.

MAJ. KREBS: It probably wouldn't work in person or at the aircraft. As you're extracting information from that flight system, ensure that the specific information requested by the research staff goes directly to the laboratory by modem and completely bypasses the flight surgeon. Changing the culture of flight surgeons is going to be very difficult.

LT. COL. DEMITRY: Perhaps the conclusion could be that a similar workshop that addresses DCS with the expertise in this room, the aircrew standards folks, and the line to resolve that issue before the first operational sorties. Perhaps we can move on.

MAJ. DIESEL: Yes. If we're using 60,000 feet as ingress and egress, that's two exposures for each mission. If your mission involves multiple target locations you will be exposed to repeated decompressions.

DR. PILMANIS: We have planned a study starting next fall on repetitive exposures. Other than a review that Dr. Sears did a few years ago, there is very little current information on repetitive decompressions. Although the review found the early data generally inconclusive, time between exposures was the most important variable, i.e., if there were only a few hours between repeated decompressions there seemed to be a higher incidence of bends while daily exposure usually resulted in the same or a lower incident rate.

MAJ. DIESEL: We have an operational example of this with the T-37 on cross country flights.

DR. PILMANIS: One point that I need to clarify. Yesterday I somewhat facetiously made the comment regarding acceptable risk that the U-2 community for 30 years has accepted what we would view as 60-80% risk, and now we're worried about an emergency short term exposure to 60,000 feet. It should be kept in mind that the U-2 pilot physiologically never sees more than 35,000 feet (40,000 feet in the earlier partial-pressure suits). Here we're talking about a pilot being exposed to 60,000 feet or higher with less than a full-coverage suit. So there is a difference.

COL. HILL: Plus the fact that the U-2 pilot prebreathes before every flight.

MAJ. KREBS: For 40 years we've been looking at compromises, e.g., the 5 PSI cockpit. We're placing more demands on our pilots as regards higher G and more protective equipment, both of which add considerable discomfort and fatigue. The newer pressurization systems are becoming more and more reliable. I think if we look past the F-22, what we need to do is consider optimizing protection for the condition where the pilot is spending most of his time. We need to seriously consider a higher cabin pressure differential to optimize performance (possibly around 7 PSI) and provide them with an environment that's less fatiguing and reduces other physiological problems.

DR. PILMANIS: I agree.

PROF. ERNSTING: I am putting up the proposed pressurization table with the operators here because I'd like to ask a question which will influence our aeromedical advice. In the current isobaric cabin, a 5 PSI differential is maintained above 8,000 feet. One of the reasons for that original design was that early on it was the area in which major air combat took place. Using this schedule, you get no changes in cabin pressure when your aircraft is flying between 8,000 and 23,000 feet. One of the reasons it has become of interest in the U.K. is that the new aircraft are now capable of maneuvering over a much greater range of altitudes. We believe that a pressurization profile that possibly begins earlier and maintains a higher differential at the higher altitudes is more acceptable. We have not yet proven it, but I suspect that it may lessen the fatigue life of the cabin. My direct question to the operators here is, will you be doing a lot of altitude maneuvering in that area outside the isobaric constant pressure envelope, i.e., between 8,000 and 23,000 feet?

MAJ. NEUBECK: In the F-22 you have an airplane that can take you higher and faster. I agree with you that the 5,000 to 25,000 foot range is where a lot of the fight will occur. The transition from the lower to higher altitudes as well as the cycle times to higher and lower altitudes will be more pronounced in this airplane. If you're going to move into that 5,000 to 25,000 foot zone where everyone else is, you really want to get in there, do your job and get away from the area. So I think there's going to be more frequent cycles through that airspace, climbing to higher altitudes, descending back down to engage and then maybe climbing back up again.

LT. COL. DEMITRY: Prof. Ernsting, you made the fatigue point from which aspect?

PROF. ERNSTING: Structural aircraft design.

LT. COL. DEMITRY: Frequent trips through these pressure ranges also seem to be very fatiguing for the aircrew. We're seeing requirements that are looking for fatigue eliminators as a force multiplier, depending on how often and what kind of smaller force we're going to have. Exposure to the hypobaric environment does seem to be more fatiguing to the pilot. Has that ever been characterized in this community?

PROF. ERNSTING: I don't know of any objective evidence for that unless the aircrew were exposed to altitudes that resulted in mild hypoxia or DCS.

DR. WEBB: An isobaric pressure from 8,000 to 23,000 feet does eliminate the need to clear the ears.

PROF. ERNSTING: When the isobaric cabin was designed in the late 1950s, much of the flight activity was in this region. In these newer airplanes, you'll be conducting aerial combat maneuvering at much higher altitudes than 25,000 feet. If you schedule the pressure slope to cut in earlier, the rate in change in cabin pressure is less for any given flight altitude. We used this argument to change the pressure schedule in the Tornado aircraft.

DR. PILMANIS: We may not be able to incorporate this concept into the F-22, but this suggestion has considerable merit and should be suggested for future aircraft design. Cabin pressurization schedules is an important issue. Are there any more comments in this area? If not, let's now discuss the likelihood of remaining at altitude following a rapid decompression. Apparently, the RAF concept is that you should descent to lower altitudes following a rapid decompression at 60,000 feet. I gather that the F-22 pilot would now like to remain at these altitudes for longer periods?

MAJ. NEUBECK: In a peacetime environment, you will immediately descend to lower altitudes. In a combat environment, if descent makes me more vulnerable to an air to air or SAM threat and I can remain a few minutes at 60,000 feet to reduce the threat, then I'll take that.

PROF. ERNSTING: I may have given the wrong impression. The driving force for partial-pressure systems in the RAF in the 1950s was to provide get-me-down protection. That's not to say that our air staff would not be interested in exactly the same extension of the system as the USAF is, in a new airplane.

DR. PILMANIS: I probably overstated the situation.

PROF. ERNSTING: Yes. If you lost cabin pressurization at high altitude in the 1950s and 1960s, in many ways the airplane was finished as a fighting weapon. You would come down immediately in this scenario.

DR. PILMANIS: Very well stated. Then, looking toward the future, there is an interest in looking at the various options for remaining at high altitude. It was suggested earlier by Dr. Ackles and some others that a 5 minute exposure at 60,000 feet is possible, using the current concept partial-pressure assembly.

In summary then, after a peacetime decompression to 60,000 feet, the safest procedure is to quickly descend as low as possible and return to base. Consideration must be given, however, to the time of exposure at 22,500 feet before loss of pressure, and exposure altitude and duration while returning to base, to determine the risk of DCS. If you were to return at very low altitudes, the DCS risk would be quite low. There is, however, a much higher risk of DCS if you remain for some time at 25,000 feet. With no prebreathing and a nominal oxygen concentration at 25,000 feet, a 50% risk of DCS can be anticipated if you are exposed for over 30 minutes on return to base.

MAJ. CAULKINS: The use of the term "risk" can be misleading, i.e., the probability of the occurrence must be considered as well as the consequences of that occurrence. Although the probability of DCS may be high, the consequences or severity of the risk may be acceptable.

DR. PILMANIS: You're absolutely right. A knee pain is not a major problem and that's what generally happens. The anonymous survey of U-2 pilots indicated a 34% decrement in performance. There is a mission impact, but not at a level that would be considered life threatening. At 25,000 feet, you're primarily going to be dealing with DCS.

MS. MCGARVEY: That is the recommended profile for F-22.

DR. PILMANIS: 25,000 feet after a rapid decompression?

MS. MCGARVEY: Yes. After a loss of pressurization. We need to remain as high as possible to provide cooling for the avionics.

DR. PILMANIS: The word to be underlined is time. How long were you exposed to 22,500 feet prior to loss of pressure and how long will you be exposed to 25,000 on the return to base?

MS. MCGARVEY: If you come down to lower altitudes, you may lose the avionics, so the time required will probably be as long as it takes to get back home.

LT. COL. DEMITRY: What you're seeing in the biomodel is that a few thousand feet really matters. As regards avionics cooling, I know that when the engineers deal with temperature deltas, 25,000 feet is a nice round number. From an avionics cooling standpoint, does the difference between temperatures at 20,000 and 25,000 feet make a large difference? What appears to be happening is that you're increasing one probability enormously at the expense of another probability. The answer may be as simple as turning off non-essential equipment? If you can return to base at 20,000 feet, you may be able to lower the DCS risk significantly? Maybe a compromise can be agreed, if the altitude is reduced by 5,000 feet.

MS. MCGARVEY: 25,000 feet was the compromise.

LT. COL. DEMITRY: Okay.

MS. MCGARVEY: The engineers really wanted the altitude to be 35,000 feet.

DR. PILMANIS: Then I would strongly suggest that they breathe a high oxygen concentration at 25,000 feet on the way back to base.

MS. MCGARVEY: The system will be driven in that direction.

PROF. ERNSTING: From the RAF point of view, coming down to 25,000 feet has always been our teaching. From an aeromedical standpoint, that is the maximum altitude at which you should return to base and it's in pilots' notes and flight reference cards for most airplanes. On the other hand, the requirement in the Eurofighter is for return at 35,000 feet, which causes us concern.

DR. WEBB: Procedures for loss of cabin pressure are contained in AFI 11-206, which is the new 60-16 for USAF pilots. It states that "if the aircraft loses cabin pressure the pilot must initiate an immediate descent to the lowest practical altitude, preferably below 18,000 feet, but in no case allow cabin altitude to remain above 25,000 feet, unless occupants are wearing functional pressure suits". The definition of a, "functional pressure suit", would be enlightening.

LT. COL. DEMITRY: These kind of guidelines have empirically withstood the test of time, which is perhaps the best research tool we have. But now we're starting to encounter harsher initial conditions.

DR. PILMANIS: Yes. Previously you weren't exposed for long periods at these higher cabin altitudes.

MAJ. MILLER: Lets look at a hypothetical situation. You lose pressure at 60,000 feet in the F-22, and select emergency oxygen at that point. You descend to 25,000 and at some point you're going to run out of 100% oxygen.

The OBOGS gas will still provide fairly high concentrations, but you may now have to eject without an emergency oxygen supply. Will the aircrew survive the ejection from 25,000 feet without additional oxygen?

COL. HILL: There would not be a problem.

DR. PILMANIS: I can state that the DCS risk at 35,000 feet would be substantially higher. Again, the risk has a time component. Will he be there for 5 minutes or an hour?

LT. COL. DEMITRY: Can you put numbers into this high-risk equation?

DR. PILMANIS: At 35,000 feet, I would simply accept that I'm going to bend, rather than guessing whether I will or will not bend.

LT. COL. DEMITRY: That's key. I mean saying that and putting that in writing.

DR. PILMANIS: Again, if you are exposed to higher altitudes for very short time periods the risks will be reduced significantly.

DR. SEARS: If you're sitting at 22,500 feet for a long time before decompression, bubbles are forming which will expand significantly upon decompression.

DR. PILMANIS: Exactly. And they aren't likely to resolve on descent.

DR. SEARS: You will probably not have much time before experiencing DCS.

DR. PILMANIS: You would be in pretty good shape if you descend to much lower altitudes, e.g., 10,000 feet.

LT. COL. DEMITRY: As a user, I would ask you to give me a number for the amount of time that I must spend at varying altitudes to give me a certain level of risk or tell me that I will bend in a certain period of time. I would like to know the shortest amount of time expected before onset of DCS and some idea of the severity. I can accept 100% probability. Just let me know what I'm up against.

DR. PILMANIS: Unless you're a very unusual individual, you're going to have to deal with DCS upon descent to 25,000 feet after a long exposure to a cabin pressure of 22,500 feet . Either you continue descent to lower altitudes or you live with DCS.

MAJ. NEUBECK: Would you decrease your risks significantly if you prebreathe 100% or 93% OBOGS gas prior to exposure to 22,500 feet for 1.5 to 2 hours.

DR. PILMANIS: Yes, depending on the length of exposure at 22,500 feet before loss of pressure. There would be some benefit, but DCS would not be eliminated. We don't have the numbers yet, but we are currently conducting the 35,000 feet runs. We have completed about 12 flights to date.

MAJ. CAULKINS: Can you tell us the percentage of the subjects that experienced DCS at 35,000 feet with an hour and 15 minutes of prebreathe?

DR. PILMANIS: It should be noted that all of our subjects were at rest during exposure to 35,000 feet. When you add any form of exercise, you're going to see a significant increase in the DCS rate. We have found a 50% DCS rate within 3 hours in resting subjects. I expect that mild exercise would increase the rate by 30 or 40%. That is why I indicated that you should expect to bend if an RD occurs after an exposure to 22,500 feet followed by descent to 35,000 feet.

MAJ. NEUBECK: How long were they exposed to higher altitudes?

DR. PILMANIS: This study does not impose an RD to 60,000 feet. The 3 hour exposure is all at 35,000 feet.

DR. GOODMAN: This is without PPB?

DR. PILMANIS: Yes. The main advantage to descending to 35,000 feet versus 40,000 feet and above is that you aren't required to pressure breathe at the lower levels.

DR. GOODMAN: What if you added some PPB? What would the DCS risk be?

DR. PILMANIS: It shouldn't change the risk of DCS. Your primary advantage at this altitude is reducing the risk from hypoxia without the need to pressure breathe.

Another question involves the chances of DCS leading to unconsciousness? During World War II they trained thousands of personnel at 38,000 feet with no prebreathing. They experienced collapse by the hundreds; not all from DCS. Their end points for DCS were so different from ours today that the data can't really be compared. Today, we don't see collapse because we recompress our subjects at the first symptom. It should also be noted that our database is limited to 35,000 feet. I don't know what will happen or how fast it might happen at 60,000 feet.

LT. COL. DEMITRY: Can the pain from limb bends be so severe as to severely impair a pilot's performance?

DR. PILMANIS: Yes, it's possible.

MAJ. CAULKINS: We bring the subjects down from altitude before the pain becomes severe.

LT. COL. DEMITRY: I understand that. Can they titrate the pain over time?

DR. PILMANIS: The initial presentation that we see is pain and paresthesias. There will be exceptions, but you can usually expect some time following the initial pain to decide whether to descend or remain at the high altitude for a longer period of time. That kind of thing can be done, but realize that the symptoms can progress rapidly. The higher the altitude, the more rapidly they could progress. I really can't give you a better feel for the problem.

COL. SHERMAN: You're using 60,000 feet as a model. Is that what we decided upon? Would it make a significant difference in the modeling if we used 65,000 or 67,000 or 68,000 feet?

DR. PILMANIS: You would quite likely increase the risk. We have very little data for these higher altitudes and to a large extent, are speculating from data collected at lower altitudes. I realize that I'm on soft ground in some of my statements. If someone has other data, please join the fray.

DR. MOON: Dr. Dick Vann has conducted many subject exposures to 35,000 feet for NASA with a three hour prebreathe and although he's seen a lot of bends, predominantly they're fairly minor bends and I would estimate that very few of them would interfere with successful flying.

DR. PILMANIS: Three hours of prebreathing will reduce DCS risks significantly, but it's not very practical for the military. The U-2 aircrew were originally required by the CIA to prebreathe for 4 hours; when the USAF took over, prebreathe was reduced to 1 hour.

COL. WORKMAN: The aircrew would reduce it further if it were left to them.

COL. SHERMAN: Some pilots would reduce it, but I've also worked with folks who came in 15 to 20 minutes early and prebreathed an hour and 20 minutes routinely.

DR. PILMANIS: In the anonymous survey, we had one retired pilot that said that he usually bent in 1 hour at cabin altitude. He increased his prebreathe to 1.5 hours and bent again. He kept increasing his prebreathe until he found

that 2.5 hours was required to reduce his symptomatology. On his own initiative, he indicated that he prebreathed for 2.5 hours before each flight.

MAJ. CAULKINS: I'd like to venture a bit beyond the decompression sickness risk and note that with the current F-22 oxygen system, a scenario that keeps the aircrew at 35,000 or 40,000 feet following a loss of pressure at 60,000 feet would impose a risk of hypoxia. The pilot will surely activate the emergency system following loss of pressure and over a period of time will deplete his 100% emergency oxygen supply. If you then lose your OBOGS, your only option will be to rapidly descend to low altitude.

MS. MCGARVEY: If he activates his emergency oxygen, he's not going to be sitting at 40,000 feet with his OBOGS gas. He should descend to 25,000 feet and if he experiences problems with OBOGS he will descend further to a safe altitude.

MACMILLAN: The Eurofighter system has got a 200 liter auxiliary oxygen supply which generates an amber warning when activated.

DR. PILMANIS: You can also turn it off.

DR. MACMILLAN: Yes. When the remaining volume goes down to 70 liters, you get a red warning.

DR. PILMANIS: In the F-22 you cannot turn the emergency oxygen off.

MAJ. CAULKINS: Right. The volume is one hundred liters and, once activated, the pilot continues to breathe from the system until the supply is exhausted.

DR. PILMANIS: It is now time to move on the more difficult topic of "sustained operations". I understand that the word has many different meanings. I was thinking of only a few minutes at altitudes around 60,000 feet and above. Can someone think of a more descriptive term?

PROF. ERNSTING: Limited stay.

DR. PILMANIS: Limited stay, okay. At these altitudes, we are confronted with several physiological problems. If you have an adequate level of positive pressure breathing to reduce the effect of hypoxia, for the limited stay you should solve most of the potential ebullism problems. I'm not sure anyone can firmly establish whether or not ebullism would impair your function over a 5 minute or longer period at altitudes to 65,000 feet. In the current equipment assembly, most assuredly it will be hypoxia that limits your stay at 60,000 feet and above. Again, if you maintain adequate levels of pressure breathing you solve not one but two problems, hypoxia and ebullism.

COL. STORK: Should we just line through ebullism and not even consider it as a problem?

DR. PILMANIS: I don't think ignoring it is correct.

COL. STORK: Would you agree that the probability of significant risk of ebullism below 60,000 feet is exceedingly small?

DR. PILMANIS: It's small, but it has been seen at 55,000 feet. More importantly, as long as you maintain PPB and lung function, it probably won't interfere with what you're doing.

COL. STORK: We can continue to discuss it in the aeromedical community, but maybe we should drop ebullism from this operationally oriented discussion.

DR. PILMANIS: Any other thoughts on that?

COL. SHAFFSTALL: Although there is no threat to life, you will experience tearing of the eyes and vaporization which may lead to visual problems that would impair capability to fly the aircraft.

DR. PILMANIS: Yes. Most of the experience base is for 1 minute exposures and now we're considering up to 10 minute exposures. I really don't know whether there will be an operational impact from ebullism.

DR. GOODMAN: We have some data from some contract work that was done looking at performance, after decompression runs. In preliminary data, the investigator feels that the ebullism didn't impair performance. The eyes tearing and the problems with maintaining the vision after the decompression, will probably be an issue.

DR. PILMANIS: An individual at NASA who lost his hose on his pressure suit at 120,000 feet, indicated that he could feel the fluid boiling on his tongue and he had visual effects during the few seconds exposure. He has not experienced any subsequent effects.

DR. GOODMAN: We had one subject who's eyes started watering. He couldn't see a thing. He was doing a mannequin task and some other things. He just said he felt fine, but just couldn't see the task.

DR. PILMANIS: I know that Dr. Sears voiced some concern a long time ago about the effects of cold on the eye.

DR. SEARS: More specifically, drying of the cornea from the vaporization of fluid during longer exposures, especially if there was little watering of the eyes via lacrimation.

DR. GOODMAN: Our subject didn't experience tearing during ground level pressure breathing. At 70,000 feet, however, his eyes watered and he couldn't see a thing.

PROF. ERNSTING: To back up what you're saying, we conducted a lot of experimental work using a partial-pressure helmet with unsleeved jerkin and G suit for the Lightning aircraft. We routinely trained aircrew to 66,000 feet and then down at 10,000 feet a minute. For these exposures, we certainly saw no problems. In later studies, I exposed my hands in a box to 70,000 and 75,000 for 4-5 minutes to study the effects of the swelling. The hands still work all right and they worked reasonably well during the 75,000 exposure. I don't think it's a terribly limiting problem.

DR. PILMANIS: We have mentioned that earlier, years ago, U-2 pilots were exposed to 65,000 feet for 2 hours.

DR. SEARS: Of course, that was using a fully pressurized helmet.

DR. PILMANIS: Yes, but several areas of their body were really not pressurized in the capstan suit. They did not report any real problems for this 2 hour period. That's a long time.

PROF. ERNSTING: They were wearing pressure gloves, weren't they?

DR. SEARS: Yes. The unpressurized areas were the crotch, armpits and feet in the capstan suit (except for flight boots).

DR. PILMANIS: You're probably correct, Col. Stork, that we can dispense with the consideration of ebullism. However, I can only guess what will happen with bubble growth and DCS at these altitudes.

DR. GOODMAN: If the cabin pressure is lost over a longer period of time, you may be able to fly at the 60,000 foot flight altitude much longer before needing to descend. From what I'm hearing, a slower decompression is a more probable occurrence operationally than a catastrophic failure of the pressurization system.

LT. COL. DEMITRY: It will be difficult to characterize the loss of pressure. Essentially you're talking about a climb, is it going to be 5,000 or 15,000 foot per minute? Perhaps it won't be as bad as a pure vertical climb.

DR. PILMANIS: Can we all agree that there will be no real problem following loss of pressure to 60,000 feet for a period of 1 minute? On a practical basis we're already there?

PROF. ERNSTING: Yes.

DR. PILMANIS: This is an emergency situation. If you were exposed to a cabin pressure of 22,500 feet for long periods before the decompression, you probably will get DCS, but for practical purposes there should be no real problems. Let us now move to a 5 minute exposure at 60,000 feet. The assumptions are that you are breathing 100% oxygen at 70 mm Hg PPB from the emergency oxygen system or 75 mm Hg from the 93% OBOGS system. Can we make the assumption that you are being adequately oxygenated for a 5 minute period?

MAJ. NEUBECK: I think you need to look at both oxygen systems. Another point that should be considered is 70,000 feet altitudes.

PROF. ERNSTING: I suggest you use absolute intrapulmonary pressures rather than specifying PPB at various altitudes. Then you can work out your gases and what your pressure breathing level will be.

DR. PILMANIS: I don't know how many of you are aware that the Germans have built a new plane called the Strato 2C for civilian use for high-altitude atmospheric research. It's designed to fly at 80,000 feet for 80 hours at a time. Their initial approach was to keep the cabin pressure at somewhere around 8,000 feet and provide a shirt sleeve environment for 2 pilots and 2 scientists. There was concern expressed regarding loss of pressure without a pressure suit. Prof. Ernsting has had closer ties to the program and might be willing to comment.

PROF. ERNSTING: I got involved first of all as an advisor because the test pilot who is going to fly the airplane is an American, from NASA/DFRC. I trained him many years back in high-altitude pressure clothing and we have a personal relationship. Pressure clothing will be used in the critical concept airplane. Both Dr. Macmillan and I have been impressed by the Grob engineering and design of the safety aspects of the cabin. We couldn't fault the design of the systems or the safety aspects. What has been interesting is the search for pressure clothing. We found that there are vast numbers of Russian capstan partial-pressure suits sitting around very reasonably priced. I became involved looking at the integration and the performance of the system. Although I had some knowledge of Russian equipment, it's been fascinating to see the detailed design which has many of the features that we pride ourselves on, in our very advanced oxygen systems. The equipment was designed for use in a 5 PSI cabin and it's now being used at much higher differentials. Provided the funding goes on, they will fly the aircraft.

DR. PILMANIS: Let's return to the question of a 5 minute exposure at 60,000 feet, assuming adequate PPB to maintain consciousness, e.g., at 70 or 75 mm Hg. Will hypoxia pose a problem? We had some earlier nods indicating that it might be possible?

PROF. ERNSTING: I think we would be concerned at the moment. I think we need to do a bit more proving of the concept of optimum jerkin/trouser pressure ratios. We've got plenty of experience of actually pressure breathing at those levels for that period of time.

DR. PILMANIS: Pressure breathing training is also necessary.

PROF. ERNSTING: I will discuss the fine tuning of the ratios in the future research needs presentation.

DR. PILMANIS: I fully expect that you will experience DCS. However, it is unlikely that it will be incapacitating in 5 minutes.

DR. WEBB: The pressure breathing required at that level will probably mask simple limb pain, so they may not know they have DCS.

DR. PILMANIS: That's right. The conclusion here seems to be that you can handle 5 minutes at 60,000 feet with some fine tuning of the suit/vest ratios. Comments?

PROF. ERNSTING: Certainly with half hour preoxygenation and then coming straight down afterwards. We had no problem with decompression sickness experimentally in a large number of subjects during 5 minute exposures.

DR. PILMANIS: Okay.

DR. WEBB: On the other hand, if they were at 22,500 feet for around an hour and were bubbling, I think you may see more than simple limb bends within 5 minutes.

DR. PILMANIS: The cabin pressure before the loss of pressure is higher than in past experience. We are in unknown territory, but from the very small amount of data that we have at 35,000 feet and data recently collected at Farnborough at 40,000 feet , as you move to higher altitude, DCS occurs in a much shorter time. This is only conjecture at this point, but I think the concept is reasonable. Again, I can't give you numbers.

LT. COL. DEMITRY: That's okay. At least I'm training your thought processes to look for numbers. If the research isn't there, will the human use safety committee let us go to 60,000 feet and stay there for 5 minutes after exposure to 22,500 feet for an hour or more ?

DR. PILMANIS: We have not conducted studies involving 5 minute exposures to 60,000 feet. In fact, we have never conducted studies to 60,000 for a 5 minute period at Brooks AFB. I noted that in Paul Webb's pressure suit studies they were exposed to 80,000 feet for 5 minutes and the subject bent even with 3.5 hours of prebreathing. There are bits and pieces of information like that, but that's about all the data we have.

LT. COL. DEMITRY: Is it impossible to do a definitive study in today's environment?

DR. PILMANIS: I'm afraid the human use committee will require a long period of prebreathing.

LT. COL. DEMITRY: As an operator, I would rather establish whether a problem really exists on the ground in a much more controlled environment, where there is immediate access to medical care, as opposed to learning about the problem in operational flight.

DR. PILMANIS: I would be surprised if we got a protocol through for exposure to 60,000 feet for 5 minutes.

LT. COL. DEMITRY: I understand the reality of the situation.

DR. WEBB: One of our protocols allowed a 15 minute prebreathe and ascent directly to 30,000 feet. The individual had severe chokes in 47 minutes. We didn't do it again. That's the closest we've come to the answer.

LT. COL. DEMITRY: Can we really make a statement that it's not a show stopper at 5 minutes? I realize there is risk, but are we even the least bit sure of that number?

DR. PILMANIS: If it were not an emergency situation, there's no way I would remain at 60,000 feet for 5 minutes. In a wartime threat situation, I probably would accept the physiological hazard over another more immediate hazard.

CAPT. O'CONNOR: Wouldn't you also want to look at the problem from a procedural standpoint? Major Neubeck earlier noted that if you know you're going to be flying at 60,000 feet, you could perhaps change your procedures where you get an hour or more of prebreathe.

DR. PILMANIS: In operational flight on the way to the tanker?

MAJ. NEUBECK: Yes. If they were going high, they probably would be able to prebreathe for 1-2 hours inflight. We rarely think about that in the operational world because we just don't go that high currently. So this is all new.

COL. STORK: Major Neubeck, the concept of operations for an aircraft like the F-22 involves flight durations of what period of time? We are discussing the possibility of sitting at 23,000 feet cabin pressures for quite long periods.

MAJ. NEUBECK: I don't have a good answer to that. In Desert Storm they were airborne for 6 hours; going across the line, dropping bombs, protecting aircraft, completing a mission and, then coming back across the line. That's a whole different scenario from a Bosnia scenario where you hang out at 25,000 feet, waiting for orders to go drop bombs. Who is to say what the operational scenario will be in 10 years?

COL. STORK: My concern is that the research community is trying to design aircrew equipment and procedures with a very limited knowledge of future operational needs or aircraft capabilities. This is not a new problem. We need now, more than ever before, to maintain a closer relationship between the aircraft developer and operator. We're really dancing with shadows when we discuss exposure time at 60,000 feet with a 5 PSI cabin or whether the maximum altitude will be 70,000 feet.

DR. PILMANIS: I think what makes it more critical now is that we're dealing in seconds and minutes rather than hours. The exposure time becomes very critical as we go higher. It's important to know these times to establish operational limits and the numbers that operators require.

MAJ. KREBS: It is unlikely that we can project how these aircraft are going to be employed in the future. If the medical community provides human tolerance limits, over a wide range of environments, that should really help the operators. It's important to present the pilot with information outlining the specific hazard as well as the probability and severity of the risk.

MAJ. NEUBECK: We have a very good idea of the concept of operations for the F-22 aircraft. We are only unsure of the location and environment where the aircraft will be used. The threat is evolutionary.

MAJ. KREBS: Yes. We built the F-15 to fly high and now we've got an E model that flies in the mud.

MAJ. CAULKINS: I think Colonel Stork is indicating that a closer connection with the user and aircraft developer would benefit the research and development program. I fully understand the reasons why the operator will generally push the airframe in an emergency, e.g., there are good reasons for remaining at 60,000 feet for up to 5 minutes over a high-threat area. The current design of the emergency oxygen system, however, will not allow you to stay at 60,000 feet for much more than a minute in the unpressurized mode. It was designed as a get-me-down system. Until the user informs us that the requirements have changed, and that they are willing to accept some increased space and weight, we can't initiate an effort to provide a better system.

MAJ. NEUBECK: And that's not likely to happen. What we're doing in this workshop is to indicate that the aircraft has the capability to remain high and are asking how far can we stretch the human capabilities at these altitudes.

DR. PILMANIS: We are also looking beyond the F-22 or derivatives.

MAJ. CAULKINS: Increasing the interaction between the research community and the user will definitely benefit future systems.

MS. MCGARVEY: In truth, most of the missions will likely center around peacetime and a get-me-down scenario which is what we have provided. What they are asking of us now is that, if I happen to be in a wartime scenario, what risk would I be taking in remaining at a cabin altitude of 60,000 feet.

MAJ. NEUBECK: Did you say that DCS would not be incapacitating for up to 5 minutes at 60,000 feet?

DR. PILMANIS: I noted that DCS will occur, but you'll probably get away with it.

MAJ. NEUBECK: But there's risk?

DR. PILMANIS: There is definite risk, especially if you have predisposed yourself with a lengthy stay at 22,500 feet prior to the loss of pressure. It is reasonable, however, to risk a 5 minute exposure at 60,000 from a DCS standpoint. Okay, now let's move on to a 10 minute exposure at 60,000 feet.

DR. GOODMAN: I understand that it will be necessary to pressure breathe at higher levels to compensate for the 93% OBOGS gas. If we could pressure breathe at 80 or 90 mm Hg to compensate for 93% FIO2 we may be able to remain 10 minutes.

MAJ. NEUBECK: In addition to looking at the higher PPB, let's look at what we have today. I also need to know the length of time the pilot will be able to remain at 60,000 feet breathing 70 mm Hg PPB.

DR. PILMANIS: If you're limited to 70 millimeters of mercury breathing 93% oxygen at 60,000 feet, you will likely become quite hypoxic.

DR. GOODMAN: The BRAG valve could be readjusted to deliver much higher PPB.

COL. HILL: We keep hearing about the next generation of the F-22. The next generation will be a larger engine, more weapons and more gas. Unless the R&D community has placed a better life-support system on the shelf by that time, it is more than likely that the older protective assembly will be used.

DR. PILMANIS: One of the purposes of this workshop is to pass along our best estimates as regards life-support capabilities, human limitations and future requirements.

MAJ. NEUBECK: We are currently considering future F-22 derivatives. We have the opportunity to tell the planners that if they want to make the future derivative aircraft more survivable by operating at higher altitudes, they had better be looking at these kind of changes in the life-support systems. The opportunity is at hand.

COL. HILL: We still owe you the answers, but we're speculating against the baseline now. Maybe you need a document that covers other contingencies?

MAJ. NEUBECK: I'm asking for what we have now, an altitude limit of 70,000 feet.

DR. PILMANIS: It's questionable whether you should even be exposed to 70,000 feet with the current concept partial-pressure assembly. If you increase the pressure breathing to compensate for the 93% OBOGS gas, that might improve your situation.

PROF. ERNSTING: It will increase the cardiovascular stress.

SQN LDR RYLES: You can also expect to get some PPB syncope.

PROF. ERNSTING: I believe the physiology is straight forward. Once we optimize the pressure ratio in the suit, you should do all right if you are breathing 70 mm Hg PPB with 100% oxygen at 60,000 feet. We don't know yet what the OBOGS gas in F-22 is going to be. MAJ. MILLER is being rightfully careful about that until you test the system. We're going to have 100% oxygen in that situation in the Eurofighter. So I think you will have to wait until the results of your tests are finished. Then you'll know what additional positive pressure you need. Once you have determined that, someone needs to assess the penalties of increasing the PPB, e.g., cardiovascular, neck discomfort and all the other things that come from breathing at high pressures.

DR. PILMANIS: You pay a price for breathing at higher positive pressure.

PROF. ERNSTING: You do and you should not consider going to 80 mm Hg . There will be a lot of aircrew that will never accept it. I know that from practical experience.

COL. SHAFFSTALL: If you activate your emergency system you get 100% at 70 mm Hg, does that significantly change the picture?

PROF. ERNSTING: I believe so. Also the emergency oxygen system could be optimized.

DR. PILMANIS: We're not there?

PROF. ERNSTING: No, not in the F-22. We are in other airplanes.

MAJ. MILLER: But then again, you have 100 normal liters of 100% oxygen. So it's going to last quite a while at 60,000 feet.

MS. MCGARVEY: You also need to descend.

MAJ. MILLER: I agree, but the supply is going to last quite a while.

DR. PILMANIS: That's good. We have a number here.

PROF. ERNSTING: I don't think any of us will disagree with that.

DR. PILMANIS: Anything beyond that is going to be a worse situation.

PROF. ERNSTING: Yes and it needs to be explored.

COL. STORK: Major Neubeck, I think you need to flag aircrew training. There will be a significant training cost for the aircrew and we need to make sure that that's a piece of this whole package. Training for PBA is different than PPG.

DR. PILMANIS: Okay, let's now discuss the DCS for a 10 minute exposure to 60,000 feet.

DR. WEBB: I think you're going to start picking up a few cases of chokes and trying to pressure breathe with chokes could be real interesting.

PROF. ERNSTING: Can I raise a general point about the bubbles in the circulation and phenomena in the lungs? I have a simplistic attitude towards that side of decompression sickness. As far as your lung altitude and circulation is concerned, 70 mm Hg at 60,000 feet gives you lung altitude of 42,500 feet. So your circulation won't go above that absolute pressure to a lower pressure. The things we will be discussing were phenomena outside the circulation, e.g., the bends. But do you greatly increase the risk of chokes above what it would be at 42,500 feet?

DR. PILMANIS: I really don't know.

COL. STORK: I think that's a really undefined region.

DR. SEARS: A very good question.

DR. PILMANIS: I think that 10 minutes at 60,000 feet without prebreathe and predisposed by exposure to 22,500 for a period of time would present a real DCS danger. The time is very important.

DR. STOLP: I totally agree that you're going to have DCS symptoms, musculoskeletal pains. You're assuming the person will be able to breathe without any problem. These are likely interactive problems.

DR. PILMANIS: True. I was attempting to separate interactive problems, which probably is not a good idea.

DR. SEARS: One of the initial reasons for this workshop was to attempt to put together some sort of interactive matrix. Dr. Pilmanis has placed considerably more thought in this area than I and should address the concept.

DR. PILMANIS: Yes, I'll put up a graph as soon as I finish. Let me return to an exposure time of 10 minutes at 60,000 feet. I think all would agree that the hazard has risen to higher level, but I don't think it will be beyond human tolerance, especially with fine tuning of the current concept assembly and a larger emergency 100 oxygen backup system? Any comments? How about a 20 minute exposure at 60,000 feet?

DR. GOODMAN: Everything becomes speculation at this point. Some of the answers could be collected without exposing human subjects to DCS, by pressure breathing hypoxic gas mixtures for 10 and 20 minutes.

LT. COL. DEMITRY: Although I see the interest in conducting studies in this area, there are other perhaps more valued places to spend the limited research dollars. I would much rather have a sliding model that was robust in the first few minutes following loss of pressure to allow me to get quite a bit further away from the threat area and, not worry about the extended exposure periods. We are being told that remaining at 60,000 and 70,000 feet can be important operationally. I think it is very important that the operational community understand that things can be quite different every thousand feet above 50,000 feet. I feel it would more useful if the research community would provide human limits for these higher altitudes for 1 to 5 minutes rather than be concerned with 10 to 20 minute exposures.

COL. SHERMAN Would it be more reasonable to provide physiological limits for 1,000 foot increments above 60,000 feet?

DR. GOODMAN: We have found that 1.5 minutes is the maximum for our subjects at 70,000 feet. With good cardiovascular support, I would say that 30 seconds would be a safe limit.

PROF. ERNSTING: I'd be quite cautious about a 1 minute exposure to 70,000 feet. I would personally like to explore the limits above 60,000 feet. The DCIEM and ourselves don't differ in the basic understanding of the systems, but we do have a lot more experience training aircrew at 60,000 feet. We're the only Air Force that has operationally used these mask, counterpressure systems and trained large numbers of aircrew. I'd be cautious above 60,000 feet. How much higher can we go? Perhaps 65,000, but 70,000? I'm still a bit concerned, because Dr. Ackles data shows he's very near the border line. When you take large numbers of people and look at the whole biological variation as well as their interest in protective systems, you will surely find some that won't tolerate the PPB required at these altitudes. I just feel that going straight to 70,000, and indicating that it's okay for 1 minute, is pushing it a bit.

DR. GOODMAN: Dr. Ernsting is right. Most of our subjects just barely make it through 1.5 minutes and their oxygen saturation's are dropping very quickly after the first minute.

DR. PILMANIS: Then, exposure for more than 1 minute at 70,000 feet is really not practical?

COL. SHAFFSTALL: Immediate descent will be necessary.

DR. GOODMAN: There's no dwell time up there.

LT. COL. DEMITRY: So if I lose pressure at 70,000 feet, I may or may not survive. Immediate descent is still going to take me some time, leaving me well above 60,000 feet for some period.

PROF. ERNSTING: If your aircraft is meeting the MILSPEC you won't have any problem at all if your engines flame out at 70,000 feet. If you lose all ECS air, at the maximum altitude, the US MILSPEC requires the aircraft manufacturer to assure that the cabin altitude shall not exceed 35,000 feet. That's how you define your leak rates. In the UK the maximum altitude requirement is 40,000 feet and every airplane has to be tested to those requirements. I have my doubts whether older generation American airplanes actually met that requirement. Certainly our experience on F-4s didn't. I think we might get away with exposure to 65,000 feet with very fine

tuning of the pressure ensemble. I think you've got to give the pilot at least half a minute at altitude before initiating descent.

DR. MACMILLAN: I agree that at least 30 seconds will be required.

PROF. ERNSTING: The 30 second period is in all of our requirements.

DR. MACMILLAN: The other thing I think that can become a bit cloudy is that we're been addressing cabin altitude. The question may not be valid if he loses his canopy at 60,000 feet. You might have aerodynamic suction, so that even though you're at 60,000 feet flight altitude, the aerodynamic effect would take you up to nearly 70,000 feet if you lose the canopy.

DR. PILMANIS: Am I to conclude that we're reaching an upper limit with these systems between 65 and 70,000 feet?

COL. STORK: You probably have passed the limit at 70,000 feet.

DR. SEARS: I think you need to only consider 65,000 feet.

PROF. ERNSTING: That's our best guess, but it still needs some additional work.

DR. PILMANIS: Are we establishing an upper limit of 65,000 feet with the available life-support systems?

COL. STORK: We also need to provide the operators a range of options, e.g., if we were to give them a full-pressure helmet and a partial-pressure suit, then we don't need to be concerned about exposures for longer periods at even higher altitudes.

DR. PILMANIS: That's why I specifically indicated the current concept partial-pressure system with mask, vest and G-suit.

COL. STORK: We need to provide information to the operator on the spectrum of systems that could be made available. We're now discussing extending the limits for get-me-down systems. And we need to inform the operator that there is a cost for protection at 70,000 feet. We can put you in a full-pressure helmet, partial-pressure suit and you can go higher for longer periods without concern. If the operator will accept more risk because a full-pressure helmet can't be used operationally, that is another option. The operator needs information on these risk factors and the equipment options that can be made available.

COL. SHAFFSTALL: Good idea. In the reconnaissance mode you may be able to use that kind of equipment and achieve higher altitudes safely.

DR. PILMANIS: You likely will not pull high G in the reconnaissance variant aircraft. Let's move on to the inflation ratio issue. In the ATAGS type suit, does everyone agree that a lower suit to vest ratio of 3 to 1 or 2 to 1 will be adequate to maintain normal cardiovascular conditions during decompression?

COL. SHAFFSTALL: I would suggest that we look at the question a little bit more, particularly if you go to operationally fitted suits, as opposed to perfectly tailored/fitted suits on test subjects in an altitude chamber. We've seen many differences during G studies between centrifuge studies and operational use.

DR. SEARS: Can we currently lock in on a ratio?

SQN LDR GRADWELL: I think the best way of putting it is that if you have lower coverage you need a higher pressure ratio and conversely, the more coverage, the lower pressure ratio required. The problem is to establish the operationally acceptable garment. Once the garment is selected, the optimum ratio can then be determined.

DR. GOODMAN: We haven't yet conducted the rapid decompression studies. There might be some interactions we don't completely understand during the actual environmental testing. I think it's safe to say we can reduce the ratio and I agree with Sqn Ldr Gradwell's comments.

SQN LDR GRADWELL: The relationship between the ground level and high-altitude runs we conducted were quite similar. Dr. Goodman's point is well taken. When you actually come down to a specific garment, a specific coverage and a range of ratios related to that garment, you need to take the system to altitude and make comparisons. However, I would be surprised if it widely varied from the responses that you see during through the wall type pressure breathing.

MAJ. CAULKINS: In your studies using a full-coverage garment at altitude, how much difference does it make how well the suit fitted prior to the inflation?

SQN LDR GRADWELL: I think the garment should be fitted properly, whether it's used experimentally or on the line. I haven't deliberately exposed people with poorly fitting garments. I wouldn't want to do it at very high altitude. If you find a poorly fitted suit, you will likely find a poorly fitted mask. We ought to make sure that the quality of fit that we provide to the guy on the line is appropriate.

LT. COL. DEMITRY: We need a test or metric so that the aircrew will leave the life-support shop knowing that their assembly is well fitted. Your point is well taken. Another very dynamic aspect is that the population will change over the course of a few months by gaining or losing weight. So as you're collecting research data, provide fitting instructions.

DR. GOODMAN: That's very important. If the suit is extremely loose, the bladders will not provide adequate uniform pressure on the body.

LT. COL. DEMITRY: Right. The life-support personnel are highly motivated, but will need the proper fitting procedures.

COL. SHERMAN: The aircrew should also be informed that they will need their suit adjusted if they gain or lose weight to ensure that the suit is actually suitable for flying.

COL. HILL: We spent an enormous amount of money fitting ATAGS . The Technical Orders are currently being completed.

PROF. ERNSTING: As far as the Royal Air Force is concerned, from the beginning it was conveyed to the staffs and the operators that we need to fit the equipment properly We're a small Air Force so we do most of our training in one center, but we have objective criteria for fitting all the garments. They're not allowed to change the size without going through the appropriate authority. On the other hand, when you've got full-coverage anti-G suit, you should be able to design it with sufficient material in the bladder that you can cover a large amount of lacing error. I'm sure your systems does, similar to our full-coverage anti-G suit. It was earlier stated that cardiovascular responses would be normalized. You may optimize them, but never normalize them.

MS. MCGARVEY: It's critical that the user understand that the aircraft performance is designed around a specific life-support system. There's no going back to the CSU-13B/P G-suit. The direction that we're moving is that you're not going to fly in this aircraft with older equipment.

COL. HILL: I'd like to ask a question, because I think this has impact beyond the F-22. Has there been any consideration to take the F-22's life-support kit and retrofit it into the F-16 and the F-15?

MS. MCGARVEY: I think General Hinton is very interested in what the F-22 is doing. They want to create a future life-support system for the Air Force that will be consistent across the F-15, F-16 and F-22. If the equipment that comes out of the F-22 is worthwhile, and it should be considered for the F-15 and F-16, then we would move out in that direction. Am I miss-stating anything?

MAJ. NEUBECK: No. The bottom line is that we can't afford to have different life-support systems in the inventory that have the same function.

COL. HILL: That's for the good of all. That only makes sense.

DR. GOODMAN: I have some comments relative to the experimentation we conducted using different ratios in the ATAGS. In the very early studies, we probably fitted the G suits more tightly than the pilot would accept operationally. We subsequently changed our fitting procedures and the data we presented are representative of operationally fitted garments. The subjects performed normal activities in the ATAGS for about 5 hours during the experimentation. It is really important to duplicate in the laboratory what you expect operationally and when it comes to fitting the garment, it's critical. As a result, we have a lot of confidence in the ATAGS data at different ratios.

MAJ. CAULKINS: I fully agree that fitting must be operationally representative. The laboratory technicians initially fitted the RAF full-coverage anti-G suit too tightly.

DR. GOODMAN: You are likely giving increased protection that you will not get operationally.

MAJ. CAULKINS: We're certainly giving them more discomfort.

DR. PILMANIS: I would now like to move on to the subject of an operational matrix. Both Dr. Sears and I had considered the concept of reviewing all the interactive variables involved in flight at 60,000 feet and above in this meeting. It became immediately obvious that it was overly ambitious to try to develop some kind of interactive matrix during the workshop. An initial listing of issues was placed in your handout. These include Aircraft/Crew Issues (flight scenarios, cabin pressurization, human factors) , Physiological Issues (hypoxia, DCS, PPB, ebullism, trapped gas, thermal problems), and Life-Support Systems (oxygen systems, regulators, masks and pressure assemblies). If a corporate database were developed that addressed the interrelationships between the various factors, it would provide the parametric foundation for those investigators who follow us to input their research data. The matrix would also allow us to quickly answer questions from the field without overlooking important variables. Are there any comments?

LT. COL. DEMITRY: Is the scope of this just a database listing interactions, or is it a dynamic modeling capability based on empirical data?

DR. PILMANIS: The initial step in the concept would be to input all the variables and interrelationships. This would include the formulations necessary to determine the interaction, e.g., rate of decompression, potential for lung damage during the decompression, oxygen requirements following decompression, pressure breathing requirements/limits, potential for DCS, type of pressure assembly required, etc., etc. You would input the computer with the flight conditions and it would screen the database for those variables that may impact the situation. Each interactive component would be presented one at a time to ensure that the answer considers all pertinent information without conducting a lengthy library search. The database must be sufficiently robust to pick out all the critical parameters.

COL. STORK: Are you talking about a peer reviewed interactive database that is entered into a computer?

DR. SEARS: That's basically it. You input the computer with accepted aeromedical data and parametric relationships to assess the effect of selected flight conditions.

DR. PILMANIS: You would be asking the database to circumscribe the impact of this set of conditions?

DR. SEARS: Yes.

DR. PILMANIS: Which parameters are going to be limiting, what is the impact of those that aren't, and to what degree. What have I overlooked?

MAJ. KREBS: I think you're going to need something like that if we plan on simplifying life-support requirements. We haven't even discussed how to combine the current concept pressure assembly with immersion protection, night vision goggles, head mounted displays, laser visors and all those other things we're going to pile on the pilot. I don't see how we sort out all the variables easily without an interactive database similar to the concept you are discussing.

COL. SHERMAN: Is there someone here that you have in mind that's going to do this? The reason I ask is that the Air Force Management Engineering Agency just completed a similar task for physiological training that inputs many different parameters. We can change one or more parameters and it will present with the information that applies to those conditions. You can input 10 or 12 variables into a 15 page EXCEL workbook spreadsheet. I have some computer whiz kids that would take this on as a challenge.

DR. GOODMAN: They probably would use relational databases. That's the kind of thing that would work.

COL. SHERMAN: I know some folks that might become supercharged relative to this concept.

COL. HILL: The word from the Air Force leadership is that they're very much enamored with this type of approach. Our response to various conditions, and certainly our decisions on acquiring systems, is heavily into modeling. The downside is we have to remember that the acquisition community is run by engineers and they know that you can reliably predict wing fatigue using mathematical models and they place tremendous confidence in those models. They do not fully appreciate that physiological systems are not as predictable. It is quite likely that once you put the model out there, that is where the decisions will come from and no longer will we be asking the people who have been conducting the research for 40 or 50 years. However, if we can couch it in the right terms it may be quite useful and you will get support from above. Air Warrior was a program established about three years ago that met an untimely death that was based heavily on modeling physiological systems and deriving answers. The idea is good if knowledgeable individuals control its use.

LT. COL. DEMITRY: The assumptions that go into this matrix need to be very, very evident to ensure that the answer is quantifiable under a variety of conditions.

DR. PILMANIS: That's part of the development phase and, of course, you should be able to input newer information.

LT. COL. DEMITRY: That will be difficult.

COL. DIXON: We surely can find an operations research expert somewhere.

LT. COL. DEMITRY: We have a full modeling and simulation capability that's at the laboratory's disposal.

COL. STORK: I don't need to remind this gathering that the modeling complexity that you're talking about is unlike anything this group has ever taken on. As Dr. Bomar mentioned with his modeling of the respiratory system and the cardiovascular system, our technology is not very far advanced. Yes, it needs to be done, but a lifetime could be invested in the accomplishment of the task. That doesn't mean that it shouldn't be started.

DR. SEARS: The project should be phased to ensure that each step of the process would provide useful information. This approach would help ensure that everything would not be lost if time and funds became unavailable.

COL. HILL: The Navy indicated four years ago that they had modeled the respiratory system all the way from the oxygen generator to the lung. I've not seen anything lately.

LT. COL. DEMITRY: From the user community, we would like the product, clearly, because the designers and the operators will all use it. The downside would be if it will require a major R&D effort for the next millennium, at the expense of providing me answers that I need in the next decade. Priorities must be balanced.

COL. STORK: The reason that there's not a life-support project for high-altitude protection is that the priority has been judged lower than ejection seat upgrades, improvements in parachute canopies and locator beacons. Everything comes from the same pot of money.

LT. COL. CLEMENT: However, we want to start looking at those kind of things. We have a priority listing that will be accomplished when funding becomes available.

DR. PILMANIS: Dr. Sears, do you have any further comments?

DR. SEARS: I have provided this group with most of the questions that have been left unanswered during the workshop. As you put the final touches on your papers , keep these and the earlier questions in mind. If you have strong opinions in any of the areas we have discussed, please incorporate them into your paper or add them as notes so that I can enter them into the workshop proceedings.

DR. PILMANIS: I would like to add that you should not view your paper or this publication as a purely academic work. As we've seen in the discussions, there are imperfect solutions to real problems, but at least we have some solutions. If you have other thoughts, please add them when you send in your paper.